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General

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MEDICAL MANUAL

OF

CHEMICAL WARFARE

FOURTH EDITION

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ADMIRALTY
WAR OFFICE
AIR MINISTRY

MEDICAL MANUAL
OF CHEMICAL WARFARE

FOURTH EDITION

1955

By Command of
their Lordships

By Command of the
Army Council

WAR OFFICE

ADMIRALTY

By Command of the
Air Council

AIR MINISTRY

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PREFACE TO FOURTH EDITION

RECENT developments in Chemical Warfare, particularly those due to the discovery of the highly toxic nerve gases at the end of World War II, and newer concepts in therapeutics and experimental physiology have rendered the third (1943) edition of the Medical Manual of Chemical Warfare out of date. It was therefore decided that a new edition should be produced.

In this new (1955) edition extensive revision has been made of the general layout and of the chapters dealing with the general description of war gases; the handling, recognition and early treatment of gas casualties; the skin irritant or blister gases; the lung irritant or choking gases; paralysant gases; arsine; anoxia and cyanosis, and the administration of oxygen and artificial respiration. A new chapter on the nerve gases has been added, and a table dealing with the effects of war gases on food and water has replaced the appendix of the previous edition.

As a new " Handbook of Occupational Health " is under preparation by the Joint Services and Factory Department Committee on Occupational Health, those subjects which could be more appropriately considered industrial hazards are not included in the revised manual. Hydrogen sulphide, ammonia and corrosive acids have therefore been omitted; but, owing to its importance in certain circumstances in war, carbon monoxide has been retained. A new note has been included on gasoline. Where accounts of gases coming more appropriately under the category of industrial hazards have been retained, these have been revised and shortened.

ACKNOWLEDGMENTS

February, 1955.

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CHAPTER I

GENERAL DESCRIPTION OF WAR GASES

Definition of War Gases

1. The term 'war gas' is applied to any substance used to reduce the fighting efficiency of an enemy by its poisonous, irritant or blistering effects.

2. The Medical Services must, however, also consider the toxic effects of various poisonous substances which may be encountered incidentally under operational conditions, but which have not been deliberately used by the enemy to produce casualties.

Historical

3. Chemical warfare, in the modern sense, was first waged by Germany in World War I when chlorine gas was successfully used on 22nd April 1915, on the Western Front. This gas was released from large cylinders in a favourable wind. The Allies were taken by surprise and as the men had no respirators casualties were heavy. Means of protection had to be improvised at once. The first official respirator (a cotton pad soaked in hyposulphite of soda, glycerine and sodium carbonate) was issued in May 1915, and after that date defence, on the whole, kept ahead of attack—so much so, that the use of phosgene gas by the Germans in December 1915 found the Allies relatively well protected against its effects.

In the hope of overcoming this protection the Germans tried arsenical irritant smokes which they hoped would penetrate the box respirator then used by the Allies. This proved a comparative failure. The use of mustard gas against the Allies was, however, highly successful.

4. Mustard gas proved to be the most effective 'war gas' used in World War I for the following reasons:—

   (a) Since it was insidious in action, had only a very faint smell and caused no immediate irritation, it was difficult to detect. There was consequently a failure on the part of the troops to realize that they were in a dangerous atmosphere which necessitated putting on the respirator.
   (b) It could attack all parts of the body not protected by a gas mask.
   (c) Ground once contaminated by mustard gas remained dangerous.

5. The main objects of using gas in World War I were:—

   (a) To produce casualties.
   (b) To harass troops by compelling them to wear respirators for long periods, thereby reducing their mobility and efficiency.
   (c) To render a selected area untenable or unsuitable for the passage of troops.
   (d) To contaminate stores, clothing and food supplies.

6. Chemical Warfare was not used in World War II and various conjectures have been made as to the reason for this. It is known that the Germans had gas available, and at the end of the war British and American forces discovered stocks of newer agents called "Nerve Gases". These were found to be effective in extremely low concentrations. Probably the high standard of training and preparedness of the Services and the fear of retaliation by the Allies were the main reasons why gas was not used.

7. The advent of the atom bomb and the fact that gas was not used in World War II do not exclude the possibility of gas being used again in war. The experiences of World War I indicate that gas is a useful strategic and tactical weapon. It can affect personnel in both forward and rear areas.

8. During World War I gas was only used in land weapons and not from aircraft; consequently gas casualties were due mainly to vapour and were largely confined to troops in the field. In a future war, gas may be dispersed by other methods on to selected targets far removed from the fighting line, such as cities, dockyards and factories. It therefore seems probable that the nature and severity of casualties may differ from those recorded in World War I.

Factors Influencing the Employment of Gas

9. The use of any gas is dependent on its physical and chemical properties and on meteorological conditions.

10. War gases may be divided into two main categories: *(a)* non-persistent and *(b)* persistent.

  (a) *Non-persistent* gases are those which remain effective for only a short time. They are released as airborne particles of a solid, droplets of a liquid, or as true gases. They are affected by prevailing weather conditions and are quickly dispersed, so that the locality in which they have been released soon ceases to be dangerous.

  (b) *Persistent* gases are liquids which evaporate slowly, giving off toxic vapours. They therefore " persist " or remain dangerous for some considerable time unless action is taken to destroy or neutralize them.

11. The following meteorological factors are likely to influence the use of gas:—

  (a) *Winds:* Strong winds rapidly disperse non-persistent volatile gases in open country, but in built-up areas, woods and dug-outs, a dangerous concentration will take longer to clear.

  (b) *Temperature:* In hot weather the least volatile and most persistent type of gas, viz., liquid mustard gas, is very effective since high vapour concentrations are obtained. In cold weather mustard gas may freeze and lie on the ground for weeks. It then presents only a minor hazard in the open, but it may be carried on boots into a warm building where it will vaporize and cause casualties.

  (c) *Rain:* Heavy rain lessens the danger from gas but does not abolish it.

Classification of War Gases

12. War gases are classified according to the clinical effects which result from their action on the human body:—

  (a) Convulsant or nerve gases.
  (b) Skin irritant or blister gases.
  (c) Lung irritant or choking gases.
  (d) Lacrimatory or tear gases.
  (e) Irritant smokes or nose gases.
  (f) Paralysant gases.

13. *The convulsant or nerve gases.* Nerve gases rapidly penetrate all moist or mucous surfaces; the vapour is quickly absorbed by the upper respiratory tract, and the cornea is permeable. In liquid form they may be absorbed through the skin. They are almost odourless, effective in low concentrations and difficult to detect. They act with extreme rapidity and when the dosage is heavy cause convulsions and death.

In small doses, headache, contraction of the pupils of the eyes, running of the nose, and tightness of the chest appear shortly after exposure. In large doses, the onset of these symptoms is followed by muscle twitching, convulsions and possibly death. If the dose is very large, death may occur in a matter of minutes.

14. *Skin irritant or blister gases.* These are compounds which in both the liquid and the vapour state may cause irritation and burning, to a varying degree, of those parts of the body with which they come in contact. The conjunctiva and cornea of the eye are most sensitive to this action and, next, the respiratory tract; the skin is somewhat more resistant.

The respirator gives full protection to the eyes and lungs, but special clothing is required to protect the skin.

Mustard gas has only a faint smell and does not cause any immediate irritation in the eyes, lungs or skin, even in dangerously high vapour concentrations. The tissue destruction produced is not usually visible for some hours and is seldom extensive. These insidious qualities make it a very dangerous gas.

15. *Lung irritant or choking gases.* The object of using this type of war gas is to cause asphyxia.

These gases are irritants which exert their main action on the lungs and the respiratory passages. In the absence of a respirator, they usually cause acute pulmonary oedema. All members of the group are obvious to the senses in concentrations which can be breathed without danger. Personnel trained in the use of the respirator should have little to fear, but if these gases were used against an unprotected or untrained civil population heavy casualties would be inevitable.

16. *Lacrimatory or tear gases.* Although many gases affect the eyes, certain compounds in particularly low concentrations have a pronounced and immediate action, causing profuse lacrimation and intense spasm of the eyelids. The concentrations required to produce these effects in the field do not harm the respiratory tract. There are some dangerous gases which besides causing lacrimation have other more serious effects, and they are not included with the relatively harmless compounds which are used for their lacrimatory qualities alone.

Eye symptoms pass off quickly when a respirator is put on. As lacrimators merely harass by forcing troops to wear their respirators, it is problematical whether this group of gases will be used against troops in future war. Their role now appears to be limited to riot control.

17. *Irritant smokes or nose gases.* These agents are usually disseminated as an extremely fine smoke or dust. They have a very faint smell. After a delayed action of a few minutes, they cause severe pain in the nose and chest, with sneezing, coughing and lacrimation. Though these distressing symptoms pass off within a few minutes of putting on the respirator, they may become worse for a short period *after* doing so.

In the concentrations encountered in the field, they do not damage the lungs and if used against trained personnel should cause only a transitory discomfort but not casualties requiring evacuation. Their future employment in war seems doubtful.

18. *Paralysant gases.* These are agents which produce their effects after absorption into the body, causing little or no local injury. The most important are hydrocyanic acid (prussic acid) and cyanogen chloride.

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(a) *Hydrocyanic acid (prussic acid)*. At high concentrations death occurs rapidly through paralysis of the respiratory centre. Although this highly toxic gas did not prove a success in World War I, it constitutes a potential danger if encountered in certain circumstances, such as in enclosed spaces. It is very doubtful whether this substance could normally be used as a war gas.

(b) At high concentrations *cyanogen chloride* has a similar action to that of hydrocyanic acid. It is not lethal at lower concentrations but has then both tear and choking effects.

Other Dangerous Gases

19. There are certain other substances, not essentially war gases, which by their poisonous action can give rise to casualties, who may need to be differentiated from casualties due to war gases. The more important members of this group are:—

(a) Arsine.
(b) Carbon monoxide.
(c) Chlorine.
(d) Fumes associated with nitro-explosives.
(e) Fumes from fire extinguishers.
(f) Gasoline.
(g) Smokes and incendiary substances.

20. *Arsine (arseniuretted hydrogen)*. This gas exerts its poisonous action only after absorption into the body through the lungs. It causes haemolysis, haemoglobinuria, often oliguria, jaundice and anaemia associated with damage to the kidneys and liver.

21. *Carbon monoxide*. This dangerous gas is present in coal gas and exhaust fumes. It is formed also by coke or charcoal braziers, and may be encountered in bomb craters and in badly ventilated emplacements. Dangerous concentrations are never likely to occur in the open. It is invisible and has no smell. The gas produces its insidious effects through its well-known interference with the respiratory functions of the blood. *Ordinary respirators are no protection against carbon monoxide*.

22. *Chlorine*. This gas was used during World War I as a choking gas, and is now used extensively in industry and for the purification of water. It is a pale yellowish-green non-persistent gas, having a strong smell resembling that of bleach. It is less deadly than phosgene but causes more initial coughing and choking. The respirator gives complete protection.

23. *Fumes associated with nitro-explosives*. These gases are given off by burning cordite or by nitro-explosives when detonation is incomplete. The danger of poisoning from these fumes is great if high explosives of this type are burned or detonated in the absence of sufficient ventilation. This may occur in gun-pits, armoured vehicles, ships' magazines and turrets, as well as in mining and tunnelling operations.

They act as powerful and very insidious lung irritants with delayed symptoms resembling those of phosgene poisoning and it is important to remember that carbon monoxide is often present at the same time. Respirators generally afford some degree of protection against nitrous fumes but none against carbon monoxide.

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24. *Fumes encountered in fire fighting.*   When fighting fires in confined space, carbon monoxide, nitrous fumes and oxygen deficiency constitute a hazard.

Chemical fire extinguishers contain carbon tetrachloride and methyl bromide, which are toxic substances.   When carbon tetrachloride comes into contact with molten magnesium, incendiary bombs or other hot metal, it decomposes with the production of phosgene, chlorine and hydrochloric acid.

Therefore, chemical fire extinguishers containing carbon tetrachloride should on no account be used for controlling incendiary bombs, especially as the service respirator will give only partial protection against the gases evolved.

Methyl bromide in high concentrations has a profound effect on the central nervous system, producing unconsciousness, epileptiform seizures and paralysis. Owing to its rapid vaporization a toxic dose may be inhaled before the danger is appreciated.

25. *Gasoline.*   Gasoline fumes are toxic to humans in concentrations over 1 per cent unless exposure is short.   Dangerous concentrations may be encountered in tanks, wagons or compartments which contain or have contained gasoline. The service respirator affords only limited protection.   When necessary, personnel should be protected by airline respirators or oxygen apparatus.

26. *Smokes and incendiary substances.*   Smokes are non-toxic in the concentrations encountered in the field but may be dangerous in the heavy concentrations formed at the site of dispersion.   Exposure near the source or for long periods in these smokes may produce irritation of the respiratory tract which can lead to serious damage, the extent of which will vary with the individual. In confined spaces, such as in buildings, concentrations may easily develop which give rise to very serious or even fatal effects.   The respirator gives complete protection.

27. *Phosphorus*, which is used as a smoke filling, is also a dangerous incendiary substance.   The flying fragments, which ignite in air, cause burns which are multiple, deep and variable in size.   The smoke is non-toxic though irritating. *Thermite, magnesium* and its alloys, and *fuel oils* are now familiar as incendiary substances.

CHAPTER II

HANDLING, RECOGNITION AND EARLY TREATMENT OF GAS CASUALTIES

Introduction

28. A suitable introduction to this chapter is the following quotation from the " Official History of the War—Medical Services ", " Diseases of War ", Vol. II (1923), page 317; it refers to the period of the battle of Loos (25th-27th September 1915) when chlorine was being used and before mustard gas was introduced:

> " A report from the West Riding Casualty Clearing Station, which admitted 248 gas casualties, stated, ' The majority of cases of gas poisoning received at this hospital showed no sign of that condition '.   Other medical officers frankly admitted that they were so handicapped by their lack of experience of cases of gas poisoning that they were often in doubt whether they were dealing with men suffering from gas poisoning or not ".

In this chapter an attempt is made to indicate and deal with the difficulties of medical officers to whom alleged gas casualties are sent. It is essential that all medical officers should be familiar with the symptoms of gas poisoning; *first*—to make a correct diagnosis in order that proper treatment may be given at once; *second*—in order to prevent men being sent back to duty when they are in the quiescent period which may follow even severe gassing with a lethal gas such as phosgene; *third*—to prevent the evacuation as casualties of those merely suffering temporary discomfort from lacrimatory or other sensory irritant gases and *fourth*—to avoid affecting clean cases by those contaminated with liquid vesicants or nerve gases.

29. When gas has been used by the enemy it is of first importance that the fullest and earliest information should be given to medical units to facilitate the diagnosis of individual cases and to permit adequate arrangements being made for the reception of casualties.

Further, it is important that all officers commanding troops in the field should be familiar with the various war gases so that men only temporarily distressed by the effects of a gas are not sent unnecessarily to medical units.

The Handling of Contaminated Casualties

30. Although the medical officer is responsible only for the personal decontamination of individual contaminated casualties (*i.e.*, persons who are both contaminated and sick or injured) who come under his care, he should be familiar with the general principles of decontamination of personnel, equipment and materials. He may also be called upon to detect contamination of water and advise on the use of contaminated food (*see* Table II).

Satisfactory decontamination is essentially a skilled procedure, hence medical personnel employed in carrying it out must be trained for the purpose, otherwise they too will become casualties. When handling contaminated wounded, personnel must ensure that all necessary anti-gas precautions are taken.

31. Contamination may vary from a few drops, which may readily escape notice until symptoms appear, to a thorough drenching from a successful low altitude spray attack or from a bursting bomb or shell. The most serious hazards are those due to liquid contamination by the nerve and blister gases.

There is one maxim that must be remembered when handling gas contaminated wounded, *i.e.*, " It is better to be alive and blistered than dead and decontaminated ". Therefore, decontamination before evacuation should be limited to life saving measures, and treatment of wounds should be restricted to such immediate first aid as is needed to make the patient fit for further evacuation. Contaminated wounded must at all times be separated from uncontaminated wounded, until decontamination has been carried out.

32. *Immediate first aid measures.* In the event of a presumably toxic liquid being splashed on the skin, there may be some doubt as to whether a man has been contaminated with liquid nerve or mustard gas. Immediate self-help and personal decontamination is the all important factor in reducing the number of casualties, and preventing the development of serious injury. If an individual is unable to help himself on account of an injury, or for any other reason, the man nearest should assist him providing the military situation permits.

When dealing with a wounded man who has been splashed with a presumably toxic liquid, there are four first aid procedures which must be adopted to prevent the development of more serious injury, or even death. These are:—

Administration of atropine, if the presence of nerve gas is suspected.

Control of massive haemorrhage.

Decontamination of the face when necessary and adjustment of the respirator.

Application of a tourniquet to a limb heavily contaminated with suspected nerve gas.

The tourniquet should be sufficiently tightly applied to obstruct the venous return, but not the arterial circulation.

The priority of these measures must of necessity depend on the clinical condition of the individual and the prevailing military situation.

33. *Evacuation.* When the patient is to be evacuated on a stretcher, precautions must be taken to prevent the stretcher from becoming contaminated by liquid gas. In World War II provision was made for the issue of anti-gas stretcher covers. These were made of laminated paper. Special oilskin sleeves were available to protect the handles.

Before use, the stretcher is placed on clean ground, opened, and then covered with an anti-gas stretcher cover. The patient, after receiving the first aid treatment described in para. 32, is lifted on to the stretcher and evacuated in the normal way. It may be found possible to avoid further harm to the patient by removing or cutting out parts of liquid contaminated outer clothing before evacuation. After thorough decontamination, further treatment of the casualty will be carried out in medical centres on the line of evacuation.

34. *Decontamination of wounded casualties.* When adequate supplies of water are available and the skin can be speedily and thoroughly cleansed, washing is an effective means of removing the liquid forms of both nerve and mustard gases. If quickly carried out there is in fact no better method for dealing with nerve gas contamination: any liquid on the skin is speedily removed by wet swabs and treatment continued by thorough washing with plenty of water and soap. Service personnel are provided with anti-gas ointment for vesicant decontamination. Both water and anti-gas ointment should be used if the nature of the gas is uncertain.

35. *Care of stretchers.* If a stretcher is splashed with liquid mustard gas it is difficult to obtain complete decontamination since the canvas is fixed to the woodwork by a large number of nails, which renders the removal and replacement of the canvas impracticable under field conditions. It is therefore important to prevent contamination. Stretchers, when not actually being used, should, therefore, be kept under cover (*e.g.*, indoors or under any improvised roof, tarpaulin, etc.). If an anti-gas stretcher cover is not available, the stretcher canvas may be protected with an improvised cover of resistant material such as a ground sheet or anti-gas cape. Such improvised covers can be decontaminated after use by one of the standard methods.

If, in spite of precautions, contamination does occur, the complete stretcher should be scrubbed with bleach paste and later washed thoroughly with water. It should then be left to weather for as long as possible. This procedure does, however, damage the canvas. The handles may not be safe for contact with the bare hands for several days, but this difficulty can be overcome by wearing anti-gas gloves. Any danger from residual contamination on the canvas can be counteracted by the use of the covers. Anti-gas ointment well rubbed into the hands, may be used prophylactically when handling contaminated material or casualties.

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Stretchers which have been exposed to the vapour of liquid gases should be allowed to weather in the open air.

History of the Case

36. The patient may be able to give a definite history of exposure to gas but his statements are often vague and unreliable. The medical officer's questions should be directed on the following lines:—

(a) Under what conditions did exposure to the gas take place? (e.g., position in the field, whether exercising actively or at rest, whether in the open or in a dug-out, etc.).
(b) What did the gas smell like?*
(c) For how long (minutes or hours) was the gas breathed?
(d) What effects did it produce at the time?
(e) Did symptoms persist after adjustment of the gas-mask?
(f) What symptoms appeared later?
(g) What period of time has elapsed since gassing—hours or days?

The replies to these questions will be of value in assessing prognosis and lines of treatment.

Recognition and Early Treatment

37. *Convulsant or Nerve Gases.*—Nerve gases are almost odourless and the vapour invisible. They have no immediate irritant effects either on the eyes, skin or respiratory tract. Therefore no warning of their presence is given to the subject exposed to them.

(a) Signs and Symptoms: In mild cases the main respiratory symptom will be tightness of the chest and throat. There will be intense headache, constriction of the pupils and difficulty of near vision. After the skin has been contaminated, nausea, salivation and vomiting may be the first signs of poisoning. Dizziness, abdominal pain and diarrhoea may also occur. These symptoms may commence several hours after exposure and may persist for days if untreated.

A *severe case* of nerve gas poisoning will present itself as one of acute asphyxia. The subject may be unconscious from anoxia, with cyanosis, sweating and pallor. Breathing will be laboured and wheezing in character, similar to that of an acute asthmatic attack. While conscious, the subject will be anxious and suffering from air-hunger; there may be restlessness and lack of co-operation due to the effects of the anoxia. Muscle fibrillation, tremors and even convulsions may be present.

(b) Treatment: To be of use this must be immediate, because of the danger from anoxia. Atropine is the antidote to the nerve gases. If there is severe respiratory distress and cyanosis, artificial respiration must be started at once and maintained until adequate respiration returns of its own accord. The portable bellows resuscitator (*see* para. 269) has been designed for this purpose and is more effective than manual methods of artificial respiration. Of the latter, the most efficient is that of Holger Nielsen, but it may be impracticable to use this method in the presence of clonic convulsions.

Subjects suffering from nerve gas poisoning tolerate large doses of atropine. An initial dose of 2 mg. (1/32 gr.) should be given intramuscularly. This dose should then be repeated—by either the intravenous or intramuscular route—until such time as the symptoms of poisoning disappear.

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*Smell of Gas.—In World War 1 descriptions by troops of the smell of gas were found to be unreliable. This was particularly true for mustard gas and phosgene and would be still more so for nerve gas. Mustard was frequently missed and it seems doubtful whether nerve gas would be recognized at all during the heat of battle because smell is, in the main, a subsidiary sense readily over-ridden. Moreover the smell of nerve gas is very faint.

38. *Mustard gas vapour.*—The characteristic symptoms are:—conjunctivitis, laryngitis, and skin burning; but these develop only after some hours.

Should a respirator have been worn during exposure, reddening and even vesication of the skin in the more sensitive regions may have occurred without damage to the eyes or respiratory tract.

The immediate treatment of such cases is the removal of the clothing which is impregnated with the vapour of mustard gas. Anti-gas ointment must never be used on the skin after reddening or blistering have appeared.

39. *Arsenical blistering vapours.*—Lewisite and other arsenical vapours irritate the respiratory tract and personnel will probably adjust their respirators before the eyes or lungs are damaged. High concentrations may cause erythema. The eyes will require treatment with BAL ointment.

The clothing of persons exposed to lewisite vapour will, unlike clothing worn in mustard gas vapour, generally have a strong unpleasant smell. Clothing smelling of lewisite should be removed as early as possible.

40. *Blistering liquids.*—The preventive treatment necessary for cases of gross contamination should have been carried out in the unit immediately after contamination, namely, removal of the clothing and application of anti-gas ointment to the affected areas of skin, followed if possible by washing with water. BAL ointment rubbed into the skin may be used in the treatment of casualties due to arsenical vesicants, if used before blistering appears. In addition, intramuscular injections of BAL may be given to assist the elimination of arsenic from the body. To be of value in the prevention of eye damage due to arsenical vesicants, the BAL ointment must be used within 10 minutes. Speed is vital.

41. *Lung irritant gases.*—The delayed action of the lung irritants introduces an uncertain factor. In doubtful cases the patient's own account is of great importance for he may describe the choking character of the gas, the cough and tightness of the chest, and there may have been definite objective symptoms such as vomiting. He may have been unduly exhausted by trying to walk to the Aid Post, and have had to be helped down; evidence in support of this may possibly be furnished by the pallor of his face and by a rapid pulse. Examination should be made to see if there is any lacrimation, any sign of cough, or unduly short and rapid breathing. It should be ascertained whether he can take a deep breath without discomfort or coughing.

The benefit of the doubt must be given to the patient but if no objective symptoms have arisen within 48 hours the gassing must have been very slight, and he can be returned to duty with little delay.

All gas cases must be evacuated as soon as possible to the point at which arrangements have been made for effective treatment. Rest and warmth are essential. Care should be taken that while waiting for evacuation, the men do not undertake any undue muscular exertion. All except the slightest cases should, as far as possible, be evacuated lying down, and walking cases should be given every assistance; they should not carry their own equipment. If any walking case shows increasing symptoms (breathlessness, palpitation, loss of power in the limbs, or a feeling of being " done in ") arrangements should at once be made to carry him.

Should pulmonary oedema develop, with accompanying cyanosis and frothy expectoration, the essential treatment is oxygen administration (*see* Chapter XIV).

42. *Paralysant gases.*—Hydrocyanic acid gas and cyanogen chloride gas in high concentration paralyse the respiratory centre, quickly producing dyspnoea, unconsciousness, and failure of respiration. In weak concentrations they are not harmless, but in comparison with other gases their action is mild: cyanogen chloride also has marked choking and lacrimatory effects.

The recognition of casualties caused by these gases presents no difficulties; a history of exposure to one or other of them, followed by great dyspnoea, unconsciousness and cessation of breathing, with or without convulsions, will be sufficient. Treatment, to be effective, must be prompt; it consists in immediate artificial respiration, combined with the administration of oxygen if available.

As antidotes, the inhalation of amyl nitrite, and the intravenous administration of 10 ml. of 3 per cent. sodium nitrite and 50 ml. of 25 per cent sodium thiosulphate may be tried; if this treatment is ineffective, 1·5 gm. of cobalt acetate in 20 ml. water may be given intravenously.

43. *Arseniuretted hydrogen* (*arsine*).—The outstanding symptoms are: haemo-globinuria, shivering, nausea, vomiting, headache, renal and perhaps hepatic pain, weakness and at a later stage jaundice and anaemia. The gas exerts no irritant action on the eyes or respiratory tract.

Treatment in the early stages will involve measures to promote diuresis, and, for severe anaemia due to the haemolysis, blood transfusion. Intra-muscular injections of BAL may assist the elimination of arsenic from the body.

44. *Lacrimatory gases.*—These are easily recognizable by the *immediate* and severe lacrimation and the spasm of the eyelids that they produce.

As met with under war conditions they are otherwise relatively harmless, and personnel against whom they are used do not usually require any treatment whatsoever. Their effects are transitory except in the unusual event of a drop of actual liquid reaching the eye, as might happen to those close to a bursting bomb or shell. The treatment of eyes so affected should consist in copious and immediate lavage, followed by such other symptomatic treatment as may be called for. The condition is seldom serious and clears up quickly, in sharp contrast with that resulting from eye contamination by drops of a liquid vesicant.

45. *Nose irritant gases.*—With these gases, the severity of the symptoms which may follow within a few minutes after inhalation may mislead an inexperienced person. The subject may look very distressed and usually complains of intense pain in the naso-pharynx, throat, and chest, with aching of the gums and teeth; coughing, sneezing, profuse salivation and expectoration are generally marked, while retching, or even vomiting, may be present.

Such a patient is only too well aware that he has been acutely poisoned by gas and may even think that he is in danger of losing his life. The treatment consists of a brief rest coupled with the assurance that recovery will be rapid and complete.

46. *Carbon monoxide.*—The recognition by a medical officer of cases of carbon monoxide poisoning in war time should not be difficult if he be familiar with the possible sources of such poisoning (as detailed in Chapter X) and if he can obtain a history.

In the milder cases the symptoms are headache, giddiness, breathlessness on exertion, weakness of the legs and incoordination of movement, mental irritability or confusion. In the severer cases unconsciousness will be profound. A blood examination may help confirm the diagnosis.

The immediate treatment should be—removal to fresh air, the administration of oxygen (if the apparatus is available), and artificial respiration if the breathing shows signs of failure. Care should be taken to keep the patient at rest and warm, and to prevent him from accidentally injuring himself while mentally confused.

47. *Chlorine.*—This gas is recognized by its greenish colour and characteristic odour of bleach. It produces symptoms of irritation to the mucous membrane of the upper respiratory passages, often manifested by a violent paroxysmal cough.

Treatment is the same as that applied to cases of poisoning by the lung irritant gases.

48. *Fumes encountered in fire-fighting.*—Casualties occurring in fire-fighting may be due to the vapours of the substances used in chemical extinguishers, to oxygen lack, to products of combustion such as carbon monoxide or carbon dioxide, or to phosgene when carbon tetrachloride is sprayed on hot metal. Although the circumstances in which they occur may give an indication of their causation, it may be difficult to assess the relative importance of the presenting symptoms. The principles of first aid treatment are, however, the same in all cases. The patient should be removed as quickly as possible to fresh air, and kept warm and at rest. In severe cases artificial respiration and the administration of oxygen may be necessary.

49. *Fumes associated with nitro-explosives.*—Poisoning by these gases has an insidious onset. The incomplete detonation of a charge in a mining gallery or the breathing of vapour arising from the spilling of nitric acid on a wooden floor may evoke a little coughing which soon subsides; hours later, however, oedema of the lungs may follow and prove fatal.

The recognition of such potential casualties must to a large extent depend on the history of exposure; when such a history is established, the treatment must, as in the case of phosgene gassing, be largely that of watchful anticipation for a period of at least 24 hours. In the meantime, complete rest is imperative, and if pulmonary oedema should develop its treatment follows closely the lines laid down for phosgene poisoning.

50. *Gasoline.*—The symptoms of poisoning by gasoline vapour are not specific, but cases are usually readily recognized by the history and the characteristic smell of the vapour.

The patient should be brought into the fresh air. Gasoline-soaked clothing should be removed and the skin washed to prevent burns.

Artificial respiration may be necessary.

51. *Smokes and incendiary agents.*—The first aid treatment for personnel exposed to high concentrations of smokes such as zinc chloride is immediate removal from the dangerous atmosphere. Adrenaline should be given if severe broncho-spasm is present, and oxygen may be necessary. If systemic absorption is suspected BAL is indicated and should be given intramuscularly.

Contamination of skin or clothing by liquid smoke-producing compounds calls for the same treatment as contamination by any corrosive, namely, removal of the clothing and thorough washing of the affected areas with water.

Burning particles of phosphorus or other solid incendiary agents should be removed from skin and clothing as quickly as possible, and the affected part then immersed in water or covered with cloths soaked in water. Copper sulphate solution (1 or 2 per cent) or copper sulphate soap should be applied if available.

52-55.

CHAPTER III

CONVULSANT OR NERVE GASES

Introduction

56. At the end of World War II stocks of a new type of chemical weapon were discovered in Germany. The filling was named " TABUN " by the Germans. It was found to be an acute systemic poison active in extremely low dosage. The toxic effects were similar to those caused by eserine and di-isopropyl phosphorofluoridate (D.F.P.). It emerged that TABUN was one of a series of compounds discovered during research on insecticides. The high toxicity of the series had excited the interest of the German War Department, and work had been going on in secret since 1937. Many compounds structurally related to TABUN have since been made, some of which are even more toxic. Those members of the series which are of military importance are now included in the generic term " Nerve Gases ".

Chemical and Physical Properties

57. (a) *Appearance.*—The nerve gases are colourless liquids, and some have a faint fruity smell. Their boiling points range from 150°–250°C. In vapour form they are invisible and some are virtually odourless.

(b) *Stability.*—They are fairly soluble in water and are very slowly destroyed by hydrolysis. Hydrolysis in alkaline solution is rapid.

(c) *Persistence* varies with different members of the series. In general it may be said that all show some tendency to cling to clothing and material and some are as persistent as mustard gas. Provision for decontamination must therefore be made.

(d) *Powers of penetration.*—In the vapour phase nerve gases are readily absorbed by inhalation and by the conjunctiva. In the liquid phase they rapidly penetrate mucous membranes and, more slowly, skin. Vapour penetration of the intact skin can occur but is slow. Since both liquid and vapour pass through fabrics, clothing cannot be relied on for protection, though naturally the greater the number of layers worn the longer the agent will take to get through. Once penetration has been established, clothing, by preventing evaporation, may possibly even assist absorption by the skin.

Toxic Properties

58. The nerve gases are very toxic substances and relatively small doses either as vapour or liquid may prove fatal. Being practically without smell and non-irritating to skin and mucous membranes, they give little or no warning of their presence. Exposure of unprotected men to barely detectable concentrations of vapour will result in harassing symptoms which, in the absence of specific treatment, may persist for several days. Lethal concentrations of vapour produce death in a matter of minutes. A few drops of liquid on the bare skin may kill within half an hour.

The nerve gases can be considered to be cumulative poisons since repeated exposures to low concentrations may eventually give rise to symptoms. This is because each exposure lowers the cholinesterase content in the blood and other tissues and, as cholinesterase regeneration is slow, a subsequent exposure may decrease the cholinesterase level to the point where toxic effects are shown. For a similar reason a patient who has recently survived a high dosage of nerve gas will be more sensitive to a subsequent exposure until his tissue cholinesterase has been substantially regenerated.

Mode of Action

59. The physiological background to current views on the action of nerve gases lies in the theory of cholinergic transmission across synapses and neural end-organs. The theory applies to somatic motor nerve endings, parasympathetic nerve endings, preganglionic autonomic synapses generally, and probably some synapses in the central nervous system. Sympathetic nerve endings, except those to the sweat glands and vasodilator fibres, are excluded. When an impulse arrives at a cholinergic nerve ending acetylcholine is liberated. This substance excites the succeeding neurone or end-organ by depolarizing its surface. Transmission of the impulse is thus achieved. Recovery of the resting state depends on the destruction of acetylcholine by the enzyme cholinesterase. This destruction is normally very rapid. It must of necessity occur within the refractory period of the particular system, *e.g.*, about 2 milliseconds for striated muscle.

60. Nerve gases cause a profound and prolonged inactivation of cholinesterase. Acetylcholine therefore accumulates at synapses and nerve endings and produces a state of more or less continuous depolarization which, in general, causes first stimulation of the end-organ and then paralysis due to a block in the transmission of impulses. Both muscarine-like and nicotine-like effects occur. The former are perhaps more prominent and may include:—

(a) miosis (with headache)
(b) salivation
(c) lacrimation
(d) rhinorrhoea
(e) bronchorrhoea
(f) broncho-constriction
(g) increased peristalsis
(h) vomiting and diarrhoea
(j) involuntary micturition.

61. The hazard to life arises from the depression of respiration which fails before circulation. In this impairment of respiratory function three mechanisms may be involved, namely, obstruction to air entry by broncho-constriction and the accumulation of secretions, neuro-muscular paralysis of the respiratory muscles and especially of the diaphragm, and failure of the respiratory centre in the medulla. The latter may be the predominant factor in most cases but the other two mechanisms will be important contributory causes of the ensuing asphyxia. The function of the respiratory centre is affected directly by the nerve gas but it is also further depressed by the resulting anoxia.

Although the heart continues to beat for some time after respiration has ceased, there is bradycardia, a diminished cardiac output, a raised pulmonary arterial pressure and an increased venous pressure.

Following a lethal or near lethal dose of nerve gas, consciousness will be lost early and, in such cases, the clinical decline is rapid and features collapse, convulsions, flaccid paralysis and respiratory failure.

62. Therapy depends upon preventing the toxic effects of the accumulating acetylcholine and relieving the anoxia due to the respiratory embarrassment. The latter requires the application of artificial respiration and the former can be achieved by a drug such as atropine. Each, however, may assist the other. Thus, the broncho-constriction, the excessive secretions and the central failure, but not the muscular paralysis, can be overcome in favourable circumstances, by the administration of atropine. (The muscular paralysis seems to be temporary in nature and function gradually returns to the respiratory muscles).

In this way the respiratory function is restored and so the anoxia reduced. By contrast, the accompanying depression of the circulation may prevent the rapid and complete absorption and distribution of injected atropine. Artificial respiration will aid the return of blood to the heart and will also reduce the anoxic depression of the heart muscle and the medullary centres. This will aid the failing circulation and encourage the absorption of atropine.

If death occurs the picture at autopsy is characteristic of asphyxia.

Signs and Symptoms

63. The account which follows is based on observation of men exposed to very low concentrations of nerve gas and on reported accidents with insecticides, such as TEPP and Parathion, which have similar actions. There is little experience of the effects of large doses of nerve gas on man. A number of experimental animals have been studied, and although the wide variation in the response of different species bespeaks caution in extrapolating the results to man, the animal results agree very well with the responses seen in man.

64. Although a full range of both muscarine and nicotine effects may be expected, it would be unwise to predict dogmatically the relative prominence of the different symptoms. It is possible to indicate the phenomena most likely to be important, but further experience may well lead to changes of emphasis. It should also be remembered that almost nothing is known about individual variation in either susceptibility or response pattern and it is well known that idiosyncrasies may exist and treatment should be planned with this in mind. Acetylcholine produces many different effects and therefore the clinical picture may vary in emphasis. So it is reasonable to anticipate a similar lack of uniformity in nerve gas poisoning.

65. The action of nerve gas vapour is rapid. Concentrations of gas insufficient to kill may produce symptoms in two minutes. With higher concentrations death may occur within five minutes. *Liquid contamination* of the skin may cause symptoms in from 5 to 30 minutes. The fully developed clinical state is likely to be much the same in either gaseous or liquid poisoning, except that in the latter eye signs may be less noticeable. Cases may be grouped as *mild*, or *severe*, the former suffering harassing symptoms only, and the latter collapse or death.

66. *Mild Cases.*—The first subjective symptom is likely to be an uneasiness in breathing. It may be accompanied by a general malaise, a little running of the nose, and some coughing. At the same time constriction of the pupils will be apparent to an observer. In daylight the man may himself be conscious of a slight darkening of the field of vision, while at night there is difficulty in adapting to the dark and in seeing clearly in artificial light. Frontal headache is an accompanying symptom and this is probably the most disabling of the minor symptoms.

Although vomiting may be an early sign of poisoning, it is not necessarily a prominent feature in cases where large doses have been absorbed.

In the absence of treatment harassing symptoms pass off rather slowly. Headache and difficulty in accommodation are the most persistent and may last four or five days.

67. *Severe Cases.*—Initial symptoms are likely to be difficulty in breathing, salivation and rhinorrhoea, vomiting, muscular weakness and incoordination, abdominal pain, and perhaps involuntary passage of urine and faeces. The tempo of the onrush of symptoms is catastrophic, and most severe cases will be unconscious before they come under medical observation.

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In the earlier phase respiration may be laboured because of obstruction from broncho-constriction and fluid in the airway and at the same time jerky from incoordination and developing weakness of the diaphragm and intercostal muscles. Bubbling and frothing may be a feature. Transient fibrillation may be seen in skeletal muscles and sometimes attacks of general clonic convulsions.

At a slightly later phase flaccid paralysis becomes general and all attempt at respiration ceases. By this time broncho-constriction may well have passed off and the airway, apart from excessive secretions, be clear. On the other hand, direct poisoning of the respiratory centre may abolish respiratory effort in advance of the onset of flaccid paralysis. Whatever the proximate cause of the asphyxia, increasing cyanosis gives a clear indication of its presence.

Profound bradycardia, with a pulse rate between 30 and 50 per minute, is to be expected. The blood pressure progressively falls—an effect which becomes more marked as anoxic cardiac failure is added to the original intoxication. The combination of falling cardiac output with peripheral vasodilatation is likely to render the pulse imperceptible in many cases.

Course and Prognosis

68. The outlook depends on the amount of gas absorbed and on the promptness and efficiency with which remedial measures are undertaken. Although the nerve gases are among the most lethal of known toxic agents, life can often be saved by treatment even though several times the lethal dose has been absorbed.

The urgent anoxia diminishes as the function of the respiratory centre and muscular power return, which in most cases should be within three or four hours. Recovery may not be immediately complete, however, and there will remain a danger of hypoxia due to recurrent bouts of muscular weakness and to the bronchial secretions which the patient is too weak to expel. Late onset of pulmonary oedema with sudden collapse and death after recovery has seemed well on the way may occur at times.

69. Recovery, when it occurs, is likely to be complete in a few days, though heightened susceptibility to further exposure will persist for some weeks. In the absence of experience of large numbers of cases little can be said about complications and sequelae, but it would be wise to be prepared for some incidence of pulmonary atelectasis and infection. The possibility of occasional cases of central nervous system damage cannot be entirely ruled out. In general, however, the vast majority of those who recover from nerve gas poisoning should have no permanent disability.

Treatment

70. *Mild cases.*—The instillation of ½ to 1 per cent atropine solution or ½ per cent atropine ointment into the eyes quickly relieves the most harassing symptoms. Atropine is more effective by this route than by injection in overcoming headache and ciliary spasm. Repeated instillation may be necessary in order to keep the pupil dilated, but most of the cases in this group should be able to return to duty after treatment.

71. *Severe cases.*—The rationale of treatment is the same as in nearly all other forms of poisoning, namely, prevention of further absorption, followed by specific therapy. Great speed is the prime essential but it is difficult to prescribe a definite order of procedure. This can be decided only by the man on the spot, according to which of the two main objects appears the more urgent.

72. *Prevention of further absorption.*—Clothing contaminated with liquid must be stripped or cut off and discarded. But it is essential that every precaution must be taken to avoid spreading contamination either on the patient or to the operator himself. Liquid on the skin should be removed by free sluicing with water or by wet swabbing. Dry swabbing should be carried out only if water is not available and the utmost care should be taken to avoid rubbing, which favours the passage of liquid through the skin. Anti-gas ointment is ineffective against nerve gas, but under active service conditions it may be impossible to be certain that a vesicant is not incorporated in the liquid. If there is doubt, the ointment should be used but then only after the contaminant has been removed, otherwise the toxicity of the nerve gas is increased by preventing evaporation.

When a limb is heavily contaminated with nerve gas, a tourniquet applied sufficiently tightly to obstruct the venous return but not arterial circulation will probably prevent further absorption.

73. When vapour is still present, adjust the casualty's respirator, and as quickly as practicable remove him to a clean atmosphere. Since the respirator interferes with the efficient treatment of anoxia, it should be removed as soon as this can be done with safety.

74. *Specific Therapy.*—Under this head may be considered:—
    (a) the reversal of the muscarine-like effects by atropine;
    (b) the treatment of anoxia.

75. *Atropine.*—A number of drugs have been shown to antagonize acetylcholine. Atropine is at present the one of choice. Its use is the sheet anchor of early treatment. A 2 mg. (1/32 gr.) dose should be given intramuscularly at the earliest practicable moment. This dose may be repeated, even several times, if the required response is not obtained. Cases of nerve gas poisoning have a greatly increased tolerance for atropine and the danger of overdosage is not great.

In urgent cases intravenous atropine may be required, but time should not be wasted in trying to perform intravenous injection in very adverse circumstances, *e.g.* the patient may be in convulsions. Better results are likely to be obtained by intramuscular injection coupled with measures to relieve anoxia. If anoxia can be controlled the circulation will often recover enough for the drug to be absorbed.

Dosage of atropine should be controlled by reference to the pulse rate. The characteristic pulse of nerve gas poisoning is slow. A rising rate is evidence that atropine is beginning to take effect. When the rate is up to about 140–150 per minute it is likely that the maximum possible benefit has been obtained.

76. *Use of atropine in the tropics.*—Since atropine inhibits sweating in man extra care is required when giving the drug in tropical climates. Full atropinisation will still be necessary in nerve gas casualties but both the administration of atropine to non-gassed personnel and over-atropinisation of gassed cases should be avoided.

77. *Anoxia.*—As asphyxia will be, in most cases, the prime cause of death, the prevention of anoxia has a most important place in the scheme of treatment.

First aid means of relieving anoxia consist of direct mouth to mouth inflation, manual artificial respiration, and the use of the portable bellows

resuscitator. As air movement will be impeded in varying degrees by both broncho-constriction and excessive secretions, it is unlikely that adequate ventilation will be achieved unless both inspiratory and expiratory phases are actively assisted. Holger Nielsen's (para. 269) is the most efficient manual method from this point of view. If the arm movements prove impracticable on a patient in convulsions, a start should be made with Schafer's method, changing to Holger Nielsen's when flaccid paralysis comes on. Better ventilation can be expected from the portable bellows resuscitator, the use of which is described in para. 269. The over-riding rule is to get ventilation going at once. It would be quite wrong to delay the start of manual artificial respiration in order to seek a resuscitator. Mouth to mouth ventilation is also useful in an emergency, and if circumstances permit, the patient could also be ventilated in an iron lung.

The clinical assessment of the adequacy of ventilation depends quite simply on the patient's colour. If he is free from cyanosis, the treatment he is getting is satisfactory.

Oxygen is a valuable adjunct to treatment and may be given by any of the recognized methods (*see* Chapter XIV). Oxygen-carbon dioxide mixtures are contra-indicated. The asphyxiated patient is already in a state of hypercarbia, and further carbon dioxide can only tend to prolong unconsciousness.

78. It is probable that a proportion of patients will be in danger of asphyxiation from inhalation of salivary and bronchial secretions, and for these continuous suction will be needed concurrently with oxygen administration. In the field much can be done by mopping out the mouth and throat or even tilting the patient to aid drainage.

79. Summary

To summarize, the treatment of an acute case of nerve gas poisoning requires the following immediate actions:—

(a) Administration of 2 mg. atropine intramuscularly—then intravenously if practicable.

(b) Removal of contaminated clothing and of liquid nerve gas from the skin preferably by swilling off with as much water as possible.

(c) The application of a tourniquet to a limb heavily contaminated with nerve gas, sufficiently tightly applied to obstruct the venous return but not arterial circulation.

(d) If breathing has ceased, application of artificial respiration by direct breathing into the air passages or if this is impracticable by the Holger Nielsen or Schafer methods. When an iron lung or bellows resuscitator is available they may be used with advantage.

(e) A careful note should be made of the pulse rate. Generally speaking, a fall below 120 per minute indicates that further atropine is required.

(f) After the establishment of spontaneous respiration prolonged vigilance is necessary, as the recurrence of anoxia from blocking of the airway or weakness of the respiratory muscles may prove fatal.

80-90. *Reserved.*

SKIN IRRITANT OR " BLISTER " GASES

Introduction

91. These are substances which act primarily by damaging the skin, the mucous membranes and the subcutaneous tissues, though remote effects may also occur after absorption into the body.

They are encountered in war as liquids or vapours and their action is attributed to interference with tissue enzymes.

Mustard Gas (H) (The " Lost " or " Yellow Cross " of the Germans and the " Yperite " of the French) was first used against the Allies on the Western Front in July 1917. Of all the chemical wartime agents used, it was the greatest producer of casualties.

Lewisite, an arsenical preparation developed towards the end of World War I, is another powerful blistering agent like mustard gas; but, unlike the latter it has an immediate irritant action on the eyes, nose and respiratory tract and (in the liquid form) on the skin, and is therefore more easily detected.

Ethyldichlorarsine, used by the Germans in 1918, is not so strongly vesicant as lewisite or mustard gas, but like lewisite is irritating to the respiratory tract.

The Nitrogen Mustards constitute another group of " blister " liquids. These substances, which are used therapeutically, may also be used in warfare.

92. It is important to remember that the phenomenal development of aircraft since World War I has been the means of introducing new methods of gas dispersion. Not only has the limited range of artillery been surpassed by the use of air gas bombs, but a more widespread and almost instantaneous dissemination of liquid agents could be obtained by aircraft gas spray—factors which have greatly enhanced the possibilities of vesicants as chemical weapons and correspondingly increased the difficulties of defence against them. It should also be remembered that in any future war it is possible that the various " blister " gases may be used together or with other gases.

MUSTARD GAS

Chemical and Physical Properties

93. *(a)* Mustard gas is 2 : 2′—dichlorodiethyl sulphide whose formula is:—

$$S \begin{cases} CH_2 - CH_2 - Cl \\ CH_2 - CH_2 - Cl \end{cases}$$
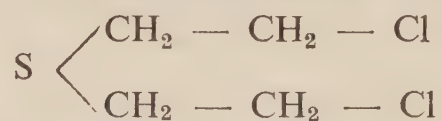
(b) Appearance.—When pure, this is a clear colourless oily liquid, boiling point 217°C., freezing point 14·4°C. The crude form is a dark coloured oily liquid. Its S.G. is 1·3 and if placed in water the bulk sinks to the bottom and the remainder spreads as a film on the surface.

(c) Odour.—Mustard gas has a characteristic odour which, though reminiscent of garlic, is faint. Thus it can readily remain undetected by those who are either unfamiliar with it or do not suspect its presence. Atmospheres containing low concentrations of mustard gas are particularly dangerous since the presence of the poison may escape detection and exposure may thus be prolonged. Further, the sense of smell tires quickly and rising concentrations in the air may thus escape notice.

(d) Solubility.—Although mustard gas is only very slightly soluble in water (under 1 per cent) both the liquid and the vapour are readily soluble in oils, fats and organic solvents generally. Thus, owing to its ready solubility in lipoids it

quickly penetrates the skin, and due to its solubility in organic solvents dissolves in alcohol, ether, gasoline, kerosene, carbon tetrachloride, etc.

(e) Stability.—Both physically and chemically, mustard gas is a stable substance; it is unaffected by normal ranges of atmospheric temperature, though simple heat disperses it by hastening evaporation. It is only very slowly hydrolysed by water; hot water, however, hastens this decomposition, the products of which (hydrochloric acid and thiodiglycol) in ordinary circumstances are practically harmless. For its chemical neutralization strong reagents are usually required, such as chlorine (as in bleaching powder), potassium permanganate, or other strong oxidizing agents.

(f) Powers of Penetration.—Both liquid and vapour readily penetrate ordinary clothing, especially woollens which contain natural fats in their fibres. Slower penetration occurs through rubber. When small drops of liquid mustard fall on clothing the injury that may result to the underlying skin is usually caused by the passage of vapour rather than by direct penetration of liquid. It sinks into all but the most impervious surfaces such as smooth metals, glass and glazed tiles.

(g) Persistence.—Liquid mustard gas vaporizes slowly at ordinary room temperatures and is therefore very persistent. Depending on weather conditions it may remain in a liquid and dangerous state for days or even weeks. It may persist under the surface of ground which appears free of the liquid. Frozen mustard gas may continue to give off vapour slowly for months. As the temperature rises the quantity of vapour given off will increase. The frozen liquid may therefore be carried by boots, etc., to warmer surroundings where it will melt and vaporize.

Methods of Dispersion

94. Weapons can be employed to exploit the offensive use of mustard gas by distributing it either in liquid form, or as an aerosol or vapour. For dispersion in liquid form it can be sprayed from tanks or containers on aircraft, by conventional bombs ground burst or air burst, and from projectiles such as artillery shell, rockets and mortar bombs. Unless these weapons function at a relatively low altitude, say 100 feet, it is necessary to thicken the charging by chemical means in order to prevent excessive losses of the agent by vaporization during its fall to the ground. In such cases the charging is of a more viscous nature, and therefore decontamination both of the body and of materials becomes more difficult. Following the dispersal of liquid mustard gas there is a danger of contact either with the liquid itself or with contaminated material, or from the evolved vapour either in the target area or downwind of it. The latter risk is intensified by high ground and atmospheric temperatures.

Sometimes the effects of mustard gas in vapour or aerosol form can be more readily achieved by the use of weapons from which the agent is distributed by means of high explosive bursts, which shatter the chargings into a fine state of dispersion.

If the temperature be sufficiently low, say 13°C. for pure material and slightly lower for production material, the agent may be encountered in solid form, and it will remain in this state for some weeks under cold conditions. On thawing out, the risks of liquid and vapour contamination are renewed.

An air spray attack on troops in the open is a possible source of danger in a future war, since the range and speed of aeroplanes enable them to produce that surprise which is so essential for the effective use of gas. Moreover, a spray attack may be launched by an enemy at such a distance from the target that the attacker can be neither seen nor heard, and the first intimation of attack may well be the arrival of the liquid spray on the troops forming the target, or even the early stages of vesication.

Under these conditions adequate defence is obviously difficult, and casualties are certain to occur even with well-trained troops. If the attack is totally unexpected a potential danger will be liquid contamination of the eyes—an occurrence that will put a man out of action for the rest of the campaign. Even if the possibility of such an attack be foreseen and personnel have their eyes protected by goggles or respirators, it will be difficult to avoid some degree of contamination of the person, and in the absence of special protective clothing mustard gas burns are inevitable unless the normal clothing be removed within a matter of minutes. The appearance of these burns will be delayed, and they will be slight or severe, localized or extensive, according to the number and size of the drops of the liquid on the clothing.

Gross contamination of the body, with correspondingly severe results, may also occur from splashes of liquid mustard gas due to proximity to a bursting mustard gas shell or bomb.

More insidious dangers from the liquid arise from the handling of material whose contamination is not suspected or from mustard gas in the frozen state.

Other Characteristics of Mustard Gas

95. (a) *Toxic properties.*—Mustard gas is a powerful cytological poison. Direct contact of the vapour, liquid or solid with any part of the body will produce local tissue damage. In addition, absorption may occur and cause various systemic effects, the haemopoietic system being particularly vulnerable to its toxic action. Cell nuclei are affected by mustard gas, and mutation may result from its effect on chromosomes. The intimate mechanisms of these actions have not been satisfactorily elucidated.

(b) *Insidious nature.*—The fact that there is no immediate irritation of the skin on contact with the liquid, nor of the eyes and respiratory tract on entering moderate concentrations of the vapour, constitutes one of the more serious dangers of this gas, as contamination may be unsuspected. Even when the gas has been detected by its characteristic odour, the sense of smell is soon dulled, or even lost, and the odour will cease to be appreciated. If, however, the respirator is speedily adjusted the odour will be detected whenever the respirator facepiece is raised to " test for gas." It is important to remember, also, that harmful concentrations of the gas can easily be masked by innocuous smokes or by fumes from high explosive.

(c) *Delayed action.*—After exposure to mustard gas vapour or contact with the liquid itself no effects are noticed for some time. Signs and symptoms may not begin to appear until after the lapse of some hours, or even days, depending on the concentration of the vapour in the atmosphere and the length of exposure thereto. By this time it is too late to ward off the effects of the gas, and casualties result.

(d) *Delayed healing.*—The affected tissues are devitalized, they are easily injured by rubbing or pressure, and they are very prone to infection. Where the gas has penetrated deeply, the healing process, even though sepsis be excluded, is very slow owing to damage to capillaries, veins and lymphatics. It is only when the action of the gas is superficial and localized that the condition clears up rapidly.

(e) *Sensitivity.*—All persons are sensitive to the action of mustard gas, and so far as is known all who have not previously been exposed to its effects possess approximately the same degree of sensitivity irrespective of race or colour.

(f) *Acquired hypersensitivity.*—In contrast to normal sensitivity it has been found that persons who have suffered injury as a result of exposure to mustard gas may in some cases become hypersensitive to its effects. The condition may be induced by either the liquid or the vapour of mustard gas. It is not possible to say with certainty whether a similar condition may be induced by other types of blister gas.

Effects of Mustard Gas Vapour

96. *(a) Signs and Symptoms*

Apart from the faint smell there are no early sensations to indicate that the atmosphere is dangerous, for the eyes, respiratory tract and skin are not irritated. This absence of immediate irritation, together with the long delay in the onset of serious damage to the body in general, make mustard gas a very insidious poison.

The eyes begin to smart and water after two or three hours and become red with a severe conjunctivitis. The nose runs with a thin mucus and there may be sneezing as in an early head cold.

Nausea, retching and vomiting associated with epigastric pain begin at about the same time as the smarting of the eyes, and may recur over several hours.

The conjunctivitis becomes gradually worse, the blood vessels deeply congested and the cornea misty. The throat feels dry and burning; the voice is hoarse, and there is a dry harsh cough.

The skin now begins to turn red on the face and neck, in the flexures of the limbs, and most markedly in the genital area.

Twenty-four hours after gassing the following appearance is typical: tears ooze from between bulging, oedematous eyelids over the reddened and slightly blistered face, and much distress is caused by headache and pain in the eyes (*see* Plate I). The combination of pain, photophobia, blepharospasm and oedematous swelling of the lids render the patient temporarily blind, apprehensive, and completely miserable. The nasal discharge, laryngitis, and cough are by now more severe but, as yet, respirations and temperature are usually normal.

Forty-eight hours after gassing the patient's condition is worse. The reddened skin is oedematous and tender, with patches of vesication. The scrotum and penis are markedly affected and cause much discomfort with blisters, swelling, and irritation. Paraphimosis may be severe.

Bronchitis now sets in with abundant expectoration of muco-pus in which may be seen sloughs from the inflamed tracheal lining.

The temperature, pulse, and respiration rates are by now raised. There is a serious danger of bronchopneumonia developing as a result of the necrosis and secondary infection of the mucous membrane of the respiratory tract. In such cases death can follow in a few days, but may be delayed for two or three weeks.

Should leucopenia develop the outlook is grave.

Rarely, abscess of the lung, bronchiectasis or even gangrene of the lung may occur, not caused directly by the vapour, but by the secondary bacterial invasion which follows. In the great majority of cases, however, the lesion is limited to a bronchitis which clears up in a month or six weeks, leaving no after effects.

(b) Pathology

(i) *Macroscopic appearances.*—The most important changes are found in the respiratory tract. From the arytenoid cartilages down to the smaller bronchioles the mucous membrane is very inflamed and covered irregularly with a thick yellowish membrane or slough; when this is removed a red granulating surface is exposed.

The lumen of the trachea and bronchi is partly filled with a thick exudate which is also seen in the smaller bronchioles on section of the lung. Oedematous areas in the lung are not a prominent feature but are generally small and localized. Small patches of emphysema may be visible.

Although the lungs are rather voluminous due to congestion, patches of emphysema and of oedema, the overall picture is quite different from that in fatal cases due to phosgene gassing where oedema is massive and the fluid drips from the lungs on sectioning.

(ii) *Microscopic appearance.*—The epithelium of the trachea and bronchi is necrosed, detached, and is seen to be composed of damaged epithelium, fibrin, leucocytes and many pathogenic organisms. Some small bronchial tubes are blocked by this inflammatory exudate and debris, resulting in localized collapse and areas of compensatory emphysema. The alveolar capillaries and the bronchioles show much congestion. The alveoli show in addition a considerable desquamation of the alveolar epithelial cells.

Patches of marked congestion in the stomach and small intestine are common. Other organs show little macroscopic or microscopic change except the bone marrow. In some cases this may show an increased production of polymorphs; in very severe cases, usually associated with an acute leucopenia, it may be pale and yellow instead of red.

It is interesting to note that in World War I mortality in mustard gas casualties was about 2·5 per cent, nearly all due to inhalation of vapour.

(c) Late effects on the respiratory tract

After World War I those soldiers who had been casualties from mustard gas, and were in receipt of a disability pension, came up for periodic examination and review. In a small but definite number of cases it was found that a progressive impairment in health was evident. The symptoms were chronic cough and breathlessness due to chronic bronchitis and fibrosis. Some had bronchiectasis. Their condition was always worse in winter. Owing to steady deterioration in the health of these men their value in the labour market progressively declined.

There is no evidence that the incidence of pulmonary tuberculosis was higher after gassing with mustard gas than among other soldiers who had no such history.

Effects of Liquid Mustard Gas

97. *General Effects.*—Owing to its high lipoid solubility mustard gas liquid rapidly penetrates the skin and the mucous membranes and causes within a few hours irritation and smarting, erythema and vesication followed by pustulation and intractable ulceration.

THE EYES

98. *(a) Signs and Symptoms.*—A splash of liquid mustard gas in the eye is painless, but severe symptoms develop in an hour or two. The dangerous effects of liquid are the same as those of high concentrations of vapour.

Intense burning pain, blepharospasm and rapid swelling of the lids develop in about 5 hours, and within 8 hours the eyes are closed. From between the swollen lids oozes a watery secretion which becomes purulent and very profuse if secondary infection develops. When the swelling subsides sufficiently for the eyes to open there is intense photophobia and the sight is misty. The cornea is hazy and oedematous and there is chemosis of the ocular conjunctiva which may protrude between the lids. The conjunctiva which has been directly exposed in the palpebral fissure may be pale and bloodless, while the surrounding portions are hyperaemic (Plate II). There is often mild iritis and in very severe cases posterior synechiae may form unless mydriatics are used. The oedema of the cornea extends through all the layers and may take 3 months to subside. Convalescence is slow and is hindered by the persistence of photophobia, lacrimation and blepharospasm for several months.

In these very severe cases vascularization of the cornea and secondary oedema set in after subsidence of the primary oedema but may be continuous with it. Such changes, occurring where there has been prolonged exposure to vapour or direct contamination by liquid mustard, are serious, for they may continue to recur for months or even years, and are accompanied by some degree of impairment of vision, which increases with each attack. Corneal ulceration and vascularization may return more than 15 years after apparent cure in cases where the scar is unstable because of the deposition of cholesterin.

(b) Complications.—The following complications occurred amongst mustard gas casualties in World War I:—

purulent conjunctivitis,
corneal ulceration,
hypopyon,
blepharitis,
styes,
Meibomian abscesses, etc.

Some of these were probably due to secondary infection of devitalized tissue. With the availability of antibiotics, etc., to combat such infections these complications may be modified in any future war.

THE SKIN

99. *(a) Effects on bare skin.*—It must be emphasized that although liquid mustard gas is a direct irritant to the skin, the reaction is not immediate. Its high lipoid solubility enables it to penetrate tissues rapidly, especially when the skin is hot; but hours may elapse before any effects appear.

The initial sign in a typical mustard gas burn is erythema at the site of contact; the capillaries are engorged, and oedema supervenes. The erythema deepens, and in severe cases may assume a livid hue. A pale, parchment-like area makes its appearance in the centre of this erythematous zone, and a tense serum-filled vesicle gradually forms (Plate III). This vesicle is the result of an inflammatory exudation of fluid which may continue for several days, according to the depth of penetration. The exudate, however, contains no actual mustard gas.

If the liquid contamination is widespread, as in a smear or splash on the skin, the erythema is followed by the appearance of numerous small vesicles which gradually coalesce to form large blebs, the underlying area being raw and oedematous; crops of such blisters may continue to develop for several days. (Plate IV). There is little or no irritation during the period of vesicle formation other than itching, especially of warm moist regions such as the scrotum. The

vesicles themselves are painless. Owing to the devitalization and impaired blood supply of the affected tissues, secondary sepsis tends to develop. Healing is slow; the resulting scar is soft and pliable and the surrounding skin often assumes a coppery pigmentation (Plate V).

Primary (neurogenic) shock is absent. If the area of blistering is considerable, some degree of haemoconcentration may occur, but the effects of mustard gas may be less serious in this respect than those of lewisite (*see* para. 116).

(b) Effects on clothed skin.—Drops of liquid mustard gas on clothed areas of the body act by virtue of the high concentration of vapour evolved, the process being assisted by the warmth of the underlying skin. Gross contamination of the clothing, on the other hand, such as may be produced by splashes or by accidental spilling, may result in actual contact of the liquid with the skin, when the action of the vapour would be superadded to that of the liquid.

All ordinary clothing is permeable to liquid mustard gas; but it is obvious that penetration will be much more rapid in the case of the single thin cotton garment of tropical and sub-tropical countries than with the multiple layers of woollen clothing worn in temperate climates. When garments are damp or wet the rate of penetration of small drops of liquid mustard gas is increased.

Protection Against Liquid Mustard Gas

100. The penetrative properties of liquid mustard gas render it necessary, for bodily protection, to adopt materials which are as far as possible impervious to the liquid. The choice of such materials is narrow, nor are they conducive to bodily comfort; hence they must take the form of additional equipment to be used when necessary.

The eyes may be safeguarded by the use of special eye shields or by the respirator. Skin can be protected by special garments, gloves and footwear.

No suitable and practicable clothing as yet discovered is completely impervious to mustard gas. Penetration by the liquid, or even by the vapour evolved therefrom, is only a question of time if no steps be taken to neutralize or remove the contaminant. *Hence the term " mustard proof" in connection with protective garments is only a relative one, and merely means that protective clothing is more resistant than ordinary garments.*

Once they become contaminated these protective materials are a potential danger, both to the wearer and to his neighbours. Special care, therefore, should be taken not to spread contamination by wearing them in enclosed spaces such as dug-outs, living-rooms, or public conveyances, nor to continue wearing them after the necessity for doing so has disappeared.

Light oilskin fabric anti-gas capes give useful protection against liquid blister gas, especially if the liquid is swabbed off at the first opportunity. Capes for stretcher-bearers have press-studs enabling them to clip the point of the cape between their legs.

Suits, anti-gas, light, are issued to special personnel and give protection for a limited period.

Protection Against Mustard Gas Vapour

101. The general remarks on protection against liquid mustard gas apply equally well to mustard gas vapour.

A well-fitting respirator will effectively protect the eyes, the respiratory tract and the skin of the face, but, in the absence of special clothing or protective equipment, the rest of the body is open to attack.

It has already been mentioned that ordinary porous clothing, especially thick, dry woollen garments, affords some measure of safety during a short period of exposure to the vapour and this protection may be greatly enhanced by suitable impregnation. The protective value is, however, removed by washing and laundering, and clothing must be subsequently re-impregnated to maintain adequate protection. It seems possible that a type of dusting powder might be made available enabling a man to protect the moist areas of the body, *e.g.*, axillae and groin. This would overcome the necessity for the re-impregnation of clothing. The objection to an oilskin cape or suit is that, owing to its stiff and impermeable character, the wearer's movements give it a bellows-like action when bending or doing manual work. Contaminated air is thus sucked inside the garment through any available channel, and with vigorous work it is only a matter of time before the atmosphere inside the suit becomes as dangerous as that outside. Where circumstances permit, the garments should be freely ventilated at frequent intervals in an atmosphere uncontaminated by the gas.

Protection against mustard gas vapour is, therefore, even more difficult to achieve than protection against the liquid.

Treatment of Injuries from Mustard Gas

RESPIRATORY TRACT

102. *(a) If the injury is mild* causing hoarseness and sore throat only, little or no treatment may be required. Codeine may be given for a troublesome cough, and a mild gargle if pharyngitis is present. Medicated steam inhalations may relieve a cough.

Should bronchitis become severe, with rising temperature and pulse rate, broncho-pneumonia may be prevented by treatment with sulphonamides, penicillin or other suitable antibiotics.

Since mustard gas may itself cause a severe leucopenia, it is particularly necessary when giving sulphonamides to carry out differential leucocyte counts. The normal response to moderate gassing by mustard gas is a polymorphonuclear leucocytosis. Should leucopenia be present sulphonamides are contra-indicated.

(b) If bronchopneumonia develops treatment follows the recognized procedures, including the use of sulphonamides and antibiotics. Venesection may be indicated at a later stage to relieve the right side of the heart of embarrassment. Oxygen is indicated only occasionally and at a late stage when cyanosis is established as a result of grave and widespread pulmonary damage.

(c) Should there be *pain in the nose* with a distressing discharge, warm douches of five per cent sodium bicarbonate solution should be applied several times daily. If mucopurulent discharge or bleeding continue because of ulceration within the nares an astringent lotion containing one grain of zinc sulphate in one ounce of boric lotion will be found helpful.

(d) Laryngeal irritation can be relieved by spraying with medicinal liquid paraffin, or by the inhalation of steam from a pint of boiling water containing a teaspoonful of a mixture of ten grains of menthol in one ounce of tinct. benzoini co. The harsh, irritating cough may be relieved by inhaling the following mixture through a Burney Yeo Mask containing a pad of gauze moistened with it hourly:—

Menthol	gr. 20
Chloroform	m. 60
Creosote	m. 60
Ol. eucalypti	m. 20
Liq. iodi mitis	m. 30
Sp. vini rect.	to one ounce

25

The menthol will help to relieve the paroxysmal coughing which may result if such an anaesthetic as simple ether has to be used for surgical treatment of accompanying injuries.

THE EYES

103. (a) *Preventive treatment.*

If protective eye-shields are properly worn droplets of mustard gas should not gain access to the eyes. If, however, they do so, the resultant injury can be lessened if the eye is immediately flushed out thoroughly with water—*e.g.*, from a water-bottle. This treatment must take precedence over other cleansing measures, such as the use of anti-gas ointment. If the eye cannot be flushed out with water within five minutes no benefit can be expected. The essential point of this treatment is that it must be *immediate*.

In view of the very serious results that may ensue from the penetration of liquid mustard gas into the eye, and the obvious difficulty of flushing out the eye immediately in the field, too much stress cannot be laid on the importance of wearing eyeshields whenever there is any risk of encountering aircraft spray.

If inflammation of the eyes is caused by prolonged exposure to the vapour of mustard gas emanating from the ground or other contaminated objects, such irrigation will not remove the mustard gas, since the poison has already been slowly absorbed and the damage done. Moreover, irrigation of the eyes takes a great deal of time and delays such essential measures as the removal of decontaminated clothing and cleansing of the skin. *The eyes should therefore not be irrigated as a routine, but only when there is a suspicion that a drop of liquid mustard gas has actually fallen into the eye within five minutes.*

(b) *Curative treatment*

The eye is the most vulnerable part of the body to injury by mustard gas.

The concentration of vapour which will cause only a minor disability to the respiratory tract, such as a mild laryngitis with mild irritation of the skin, is sufficient to cause conjunctivitis of almost crippling severity for a week or more.

The rapidity of onset of conjunctivitis depends on the concentration of vapour in the area, and the duration of exposure, varying from a few hours to two days. Once conjunctivitis is established, irrigation will have no direct effect on the development of the mustard gas injury, and therefore no time should be spent on it. The profuse flow of tears will do all that irrigation could do.

By the time the casualty is seen there is sure to be some degree of conjunctivitis, accompanied by swelling of the eyelids, and photophobia. The lids should if possible be opened to let the patient see for himself that his sight is not lost and to inspect the cornea. It should be explained that he must go through a period of increasing discomfort before improvement begins and that he will recover.

At the earliest opportunity a drop of mydriatic should be put into the eye; 0·5 per cent atropine solution is suitable for this purpose. After the mydriatic has acted it may be useful to instil a few drops of a mild antiseptic. Strong antiseptics must on no account be used, and weak sulphacetamide drops (2·5 per cent solution) are recommended.

Never irrigate unless the discharge is very copious. Experience has shown that repeated (*e.g.*, two-hourly) irrigation increases the severity of the lesion. If the discharge is excessive, wash it out with normal saline night and morning. A mild antiseptic may be instilled occasionally; but the essentials of treatment are to keep the eyelids very clean and the pupils dilated.

If blepharospasm is sufficient to close the eyelids completely, the case should be seen by an ophthalmic surgeon as soon as possible for two reasons:—

(i) So that mild cases may be diagnosed as such and not be transferred as severe casualties. This will lessen the development of " functional " cases and therefore reduce the drain on the fighting troops.

(ii) So that severe cases may be diagnosed as such and appropriate action taken.

Cocaine should not be used to relieve pain since it has a deleterious effect upon the corneal epithelium which is already damaged by mustard gas.

The eyes should neither be bandaged nor closely covered, since free drainage of the discharge is essential, but a forehead shade is permissible. Such a shade can be easily improvised from brown paper or other material.

Never instil liquid paraffin (or other oily drops) during the first few hours, but after that it may be used to prevent the lids from becoming adherent.

Many of the casualties caused by exposure to vapour are likely to be mild. None the less such casualties may at first be unable to see owing to the swelling of the eyelids and pain, and the early photophobia, which has a genuine cause, may be succeeded by a functional photophobia which unduly protracts convalescence. If this is to be avoided and the mild cases returned to full duty without needless delay it is essential to remove the eye-shade as soon as possible and to make the casualty confident that he need fear no permanent injury to the eye nor impairment of vision. General tonic treatment, and suitable exercise that will keep the casualty from brooding on his condition, will hasten his recovery.

Mustard gas has, as the experience of the first war showed, a very high casualty-producing value, and undue wastage of the fighting troops can only be prevented by taking trouble over the treatment of the milder casualties so as to minimize the time spent either in the hospital or the convalescent camp.

It is, of course, the casualties who have been exposed for a considerable time to a heavy concentration of vapour, and in particular those cases in which a drop of liquid mustard gas has entered the eye, that offer the most difficult problem, and it is these cases which demand the most careful attention of the ophthalmic specialist from the start.

THE SKIN

104. *(a) Preventive treatment*

(i) To save the skin from damage it is necessary (1) to remove any clothing contaminated by liquid or vapour*; and (2) to free the skin from contaminating liquid.

Prompt local cleansing will suffice for small localized liquid contamination of the bare hand, and a change of clothing is all that is necessary after exposure to a low concentration of the vapour. Each case will have to be considered on its own merits. But whatever the type or extent of the contamination, speed is the essence of all preventive treatment. When the skin is hot as a result of exercise, and in hot or tropical countries, the need for prompt action is especially great.

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* Should a Thomas's splint have to be applied, special care will have to be taken not to restrict the circulation through the foot, if it is necessary to remove the boot owing to its contamination by liquid mustard gas.

After exposure to vapour, or after the outer clothing has been sprayed or has come into contact with fluid, the most important measure is the removal of clothes. In deciding how much clothing must be removed it should be borne in mind that a single drop of mustard gas on a garment can cause widespread vapour burns. As an additional precaution immediate washing of the skin with soap and water is recommended, though not essential.

(ii) For contamination of the skin by liquid there is a choice of methods.

The best to adopt is the one most readily available.

(1) *Swabbing.*—If the liquid is visible it should be removed by swabbing, *e.g.*, with cotton-waste.

(2) *Anti-gas Ointment* should be applied in small quantities to the affected areas and rubbed in vigorously for not less than half a minute. Ointment neutralizes the mustard gas and should prevent blisters if applied within five minutes of contamination. It may also be useful later if there is still fluid on the skin; but where erythema has actually developed it is useless and indeed harmful. Care should be taken not to let it enter the eyes.

The ointment now used, Anti-gas Ointment No. 6, is a vanishing cream easily worked on the skin and completely absorbed by it. It will remain at good consistence over the temperature range of 10°–50°C. and hence will not deteriorate in tropical climates. The formula is as follows:—

Anti-verm	25
(2:4-dichlorophenyl benzoyl chloroimide)	
Diethyl phthalate	20
Hardened groundnut oil	10
Sodium stearate	4
Potassium stearate	2
Water	39

Anti-gas Ointment No. 6 is also a useful prophylactic if applied to the skin when exposure to mustard gas is expected. *It may be used for prophylaxis in hot, sweaty, climates, but there is a danger of the Anti-verm being absorbed through the skin, giving rise to methaemoglobinaemia.*

Old stocks of Ointment No. 5 may occasionally be encountered and are satisfactory for temperate climates only.

(3) *Aqueous bleach paste* may be used instead of ointment, and is preferable when large areas have to be covered. It can be painted on the skin with a brush, but must be washed off one minute later. Like the ointments, it should not be used after erythema has developed, and it is dangerous to the eyes.

The aqueous bleach paste consists of " supertropical " bleaching powder mixed to a creamy consistency with water—roughly, one part of the powder to one or two parts of water by volume, and it retains its effectiveness in temperate climates for several weeks if it is stored in enamelled containers with well-fitting lids. Bleaching powder is ordinary chloride of lime, while supertropical bleach is ordinary bleaching powder stabilized by the addition of quick-lime and fulfilling certain conditions of stability and chlorine content.

Ordinary bleaching powder is more irritating to the skin than the supertropical variety, but in the absence of the latter it is quite suitable for preventive treatment when made into a paste with water, provided prolonged storage is not contemplated.

(4) *Chemical solvents.—Repeated swabbing of the contaminated area with gasoline, methylated spirit, kerosene, carbon tetrachloride (Pyrene liquid) or other solvent of liquid mustard gas.* It is important to remember that these solvents do not destroy the gas, but merely dissolve it; *hence the swabbing must be confined strictly to the contaminated area.* Oilskin or rubber gloves must be used if they are available; otherwise, the swab should be only partly moistened with the solvent, and it should be held between finger and thumb by the dry portion or preferably in forceps. The wet portion is applied to the contaminated skin so as to soak up the liquid contamination, care being taken that none of the solvent runs over the skin of either the subject or operator; the contaminated swab is then discarded and the process is repeated with fresh swabs for several minutes, or as long as the characteristic odour of the gas persists on the skin. Thorough washing with soap and water, if available, will complete the treatment; the contaminated swabs must, of course, be destroyed by burning or burying and the gloves and forceps decontaminated.

One disadvantage of this method in the hands of unskilled persons is that the solvent is apt to " run " on the skin and cause burns on areas comparatively far removed from the original site of contamination; a further disadvantage is that the operating fingers are liable to become contaminated in the absence of gloves. Employed with care and intelligence, however, the method should be valuable in emergencies at aerodromes, among mechanized transport, and in other situations where mustard gas solvents are at hand. It is also a good means of removing mustard gas from burns already showing erythema.

(5) *Washing with soap and water*, using hard soap and frequent changes of water is a very effective method. This process does not destroy the mustard gas, but merely removes it in the lather; the washing must, therefore, be confined to the contaminated area, and the operator's hands should be safeguarded, if possible, by suitable gloves.

If the liquid contamination be small, localized and of known situation, this is an effective method of removing it, if carried out promptly. Vesication of the skin is usually prevented if the treatment is not delayed beyond five minutes, though an erythema may follow.

With gross contamination, or when the drops of liquid mustard gas are numerous, the results of washing with soap and water are unfavourable, as it is difficult to avoid spreading the contaminant in the soapy lather to surrounding areas. In these circumstances bleach treatment is best.

Where erythema has already begun, the use of a solvent is recommended; but if cleansing is delayed, thorough washing should still be carried out at the first available opportunity in the hope of mitigating the burning.

(b) Curative treatment

(i) *General.*—Sepsis is the most potent factor in delaying the healing of burns, and the intelligent use of antibiotics should help to reduce the seriousness of these casualties. When it is remembered that troops in the field are unavoidably dirty, and that mustard gas devitalizes the skin, it is obvious that early preventive treatment is of paramount importance.

As a preliminary to all local treatment it is essential to cleanse the skin as thoroughly as its damaged condition permits, and to clip short all hair, if any, on the affected area. *The application of bleach in any form to a skin which is already showing signs of damage will aggravate the ensuing burn.* It must also be noted that skin surfaces damaged by mustard gas are exceedingly susceptible to trauma, and that even the continued pressure of an ill-fitting bandage may extend the damage.

The clinical appearance and the pathological lesions of mustard gas burns are similar to those of normal thermal burns.

After decontamination mustard burns should therefore be treated in the same way as thermal burns, bearing in mind, however, the following points:—

The devitalization of tissues and the impaired blood supply cause delay in healing and fragile scars.

In the past sepsis proved to be a common complication and a potent factor in delaying healing of mustard burns. The intelligent use of antibiotics and early attention to asepsis should reduce this risk to a minimum.

Trauma to the affected areas must be avoided since the skin surfaces are very susceptible to injury, both physical and thermal.

The cardinal points in the treatment of burns are:—

The replacement of fluid loss.

The use of sterile burns dressings.

The use of antibiotics.

The administration of morphia in full doses where necessary.

As treatment will vary according to the nature and degree of the burns, it will be best to consider these in detail:—

(ii) *Erythema.*—Mild cases which do not proceed beyond an erythema heal spontaneously, with possibly some desquamation and pigmentation. They may be compared with sunburns in severity and discomfort, and clear up just as readily. After washing with soap and water or with Dettol (*e.g.* 4 per cent solution) a mildly antiseptic dusting powder may be applied (*e.g.*, zinc oxide, starch, boric acid and chalk in equal parts), or calamine cream. Where sepsis is anticipated the area may be treated with bacteriostatic agents, such as penicillin or D.B.P. (di-bromopropamidine isethionate) ointment. In the scrotal region irritation can be relieved with 1 per cent acriflavine or with calamine lotion containing 1 per cent carbolic acid. A warm bath is another useful means of relieving irritation.

(iii) *Vesication.*—Small blisters can be treated in the same way as erythema and may disappear without breaking. Isolated large blisters should be evacuated with a syringe under aseptic conditions or opened with a scalpel or needle, gentle pressure being applied, if necessary, upon the walls of the blister with a sterile swab to ensure complete evacuation; the intact epithelium should then be allowed to collapse and protect the raw, sensitive surface underneath. This evacuation of fluid from blisters may have to be repeated owing to the continued oozing of serum from the raw area. The further treatment of these isolated vesicles consists in the application of dry sterile dressings or saline packs.

When a considerable area is blistered, not only the lesion but also all the surrounding skin which may ultimately show damage should be cleansed with a non-irritating antiseptic such as Dettol solution, hairy parts being shaved or clipped. The blisters, unless already broken, should be punctured when they become tense. References to tanning and coagulation treatment which appeared in the third edition have been omitted, as this treatment has no place in modern therapy. The long duration of the exudation depends in part on the fact that the action of the poison continues for a considerable time after it has been absorbed by the skin, and the damage is therefore progressive. Measures which result in a reduction of the local oedema are therefore desirable.

Treatment at this stage with amyl salicylate helps to dry up the exudation, reduces the surrounding oedema and erythema, and diminishes the pain, irritation and discomfort.

In the absence of amyl salicylate, a pad of sterile gauze wrung out in sterile normal saline should be applied to the burn in the early stages, being changed as often as it becomes soaked with exudate. As soon as the exudation has diminished sufficiently, the blister skin should be removed and the area treated as for thermal burns. Crusts on the face may be removed with copper sulphate solution (copper sulphate gr. 4, zinc sulphate gr. 6, camphor water 1 oz.: dilute 1 part in 8 parts of water).

The healing of mustard gas burns is a slow process. In the case of the deeper burns a granulating area may be left, and treatment with a stimulating ointment, e.g., scarlet red ointment, may be helpful, care being taken not to damage the new granulation tissue; and early skin grafting must be considered. Preparations useful for lingering dermatitis and irritation are blue paint (brilliant green 1/1,000 and crystal violet 1/1,000 dissolved in equal parts of rectified spirit and water) and D.B.P. ointment.

Severe burns in the neighbourhood of a joint may demand early immobilization, and consideration should be given to early skin grafting.

If the burn becomes septic, or there is deep necrosis of the skin, *mildly* antiseptic baths may be useful and soothing, warm hip baths of isotonic salt solution allaying the intense irritation of mustard gas burns of the genitalia. Hot fomentations are unsatisfactory. There is nothing to contra-indicate the use of penicillin powder on the wound.

The slow progress of mustard burns under treatment, compared with thermal burns, is attributable not only to lowering of the vitality of the tissues by the poison but also to persistence of an irritative process in the tissues even when necrosis has ceased. These factors explain the greater intensity of the inflammatory reaction, the longer delay in repair, and the special tendency to septic infection.

SYSTEMIC TREATMENT

105. Where nausea, vomiting or epigastric discomfort are present, the diet should be light and fluids may be given freely, especially milk and beef tea; should these not be retained, the administration of gr. 10 to 20 of sodium bicarbonate may be of assistance, and the patient should be encouraged to drink water freely. As convalescence proceeds, and in all cases of uncomp.icated body burns, a full diet is required, and this should be as varied as possible. As with thermal burns, intramuscular penicillin should be given for the prevention of infection. If agranulocytosis occurs, whole blood transfusion and pentnucleotide may be required.

Quoting from the " Official History of the War ":—" The management of the convalescent period provides the great test for the Medical Officer's ability, because he is required not only to treat the disease, but to restore morale, to cut short hospitalization, and to lift men out of the slough of self-analysis which so often follows gassing. The best results will therefore be obtained by placing gassed cases in selected hospitals under the care of Medical Officers with special aptitude for this.

" As soon as a man is convalescent and free from the danger of septic complications, he should be discharged from hospital to a convalescent centre, where a well-ordered routine of exercise, employment, amusement and rest will quickly restore him to a state of physical and mental fitness."

Functional After-effects

106. In World War I functional disorders led to wastage and invalidism which, to some extent, could have been avoided if personnel generally, and medical officers in particular, had been more conversant with the limitations as well as the potentialities of chemical warfare substances.

Functional disorders fall in the main into two classes. In the first, exposure to gas—often to a minimal and barely a toxic concentration—may prove the final factor in upsetting a nervous system already breaking down as the result of physical or mental strain. In such circumstances, and especially when combined with ignorance, it may produce an " anxiety state."

In the second class a local, but real, organic lesion from mustard gas causes irritant reflexes, such as coughing or photophobia, and these are perpetuated by introspection, almost in a form of conversion hysteria, long after their organic cause has been cured. Lack of appreciation of this possibility by medical officers will cause much delay in returning men to duty.

Functional photophobia and aphonia are responsible for the great majority of cases. This is not surprising when it is realized that the initial trauma affects a highly organized special sense, and that fear of blindness or dumbness resulting from the injury may very well prolong the symptoms. Ill-advised and unnecessary treatment, however, is also a probable factor in many cases, as, for example, the continued retention of eye-shades long after the necessity for them has passed and the actual lesions have totally disappeared.

Persistent aphonia, often accompanied by a useless, harsh cough, is another striking example of auto-suggestion arising from the initial laryngeal irritation. The characteristic cough is either dry, or accompanied by watery sputum mainly of salivary origin. It is usually much worse at night, and is of a ringing harsh quality. If the medical officer realizes the nature of the condition and gives the patient confidence in his early recovery, this functional aphonia yields very rapidly to treatment by suggestion and breathing exercises.

Of all after-effects, functional or organic, those which seem to affect the heart present the greatest difficulty in assessment. Effort syndrome, with its shortness of breath and tachycardia following exercise, arises from so many diverse causes that gassing, in World War I, was naturally regarded as one of them. It is clear, however, that under competent medical treatment, the incidence of effort syndrome in mustard gas casualties should be very low when serious complications, such as broncho-pneumonia, have been absent.

Invalidism after Mustard Gas Poisoning

107. Experience from World War I showed that the chemical damage to the skin, to the respiratory passages, and to the outside of the eyes might cause prolonged devitalization of these tissues and a poor resistance to secondary bacterial infection, to say nothing of bone-marrow damage. There has been no subsequent evidence of the irritation leading to later malignant changes in any tissue. The trachea and bronchi in some cases showed a tendency to relapses of bronchitis, but there was no special proneness to pulmonary tuberculosis. In certain cases of severe injury to the eye, however, recovery was very slow, owing to repeated fresh ingrowths of blood vessels, followed by deposition of cholesterin, which in 15 years or less, led to corneal ulceration (delayed keratitis) after a varying symptomless period.

Invalidism in general was not prolonged, but it should be remembered that the casualties were very largely due to mustard gas vapour, and only rarely to direct splashes of the liquid. In all cases admitted to hospitals there was some degree of conjunctivitis and laryngitis as well as skin burns. The skin lesions

from vapour healed quickly, usually in less than a month. Prolongation of invalidism was due rather to trouble in the respiratory passages and eyes, and to general debility. By following up the times of recovery in a large number of cases at convalescent depots in France, it was proved that at least 75 per cent of mustard gas casualties admitted to hospitals in the lines of communication, these being the severer cases evacuated from the army zone, could be returned to full duty in less than eight weeks. This involved an average stay in hospital of two to three weeks, during the last half of which time the casualties did not require to be in ward beds or to be specially attended by nursing orderlies.

The worst cases might remain in hospital for two months or even longer. Photophobia, either functional or associated with a tendency to recurrent keratitis, often lingered. Next as causes of invalidism came bronchitis and laryngitis, and lastly effort syndrome and neurasthenia or some general debility. But out of a group of nearly 800 severe cases detained in hospital beyond the ninth week, none died and ultimately only 0·5 per cent were discharged as permanently unfit for service. The ultimate invalidism from mustard gas vapour was therefore very small. As in phosgene poisoning, it is probable that any persistent chest trouble was due to secondary bacterial infections rather than direct chemical action.

The Use of Mustard Gas in the Tropics

108. It was noted in World War I that mustard gas was much more effective in summer than in winter. Although it has not been used in tropical warfare, it is clear, both from theoretical considerations and from experimental evidence, that mustard gas is a much more potent weapon in tropical than in temperate climates for the following reasons:—

(a) The concentration of vapour evolved from weapons is much higher owing to the increased atmospheric temperature.

(b) The skin burning power of mustard gas is much increased because of the greater receptivity of the skin in tropical climates. Thus, extremely low atmospheric concentrations may cause scrotal burns, etc. As an illustration of the increased absorption, it has been shown that when liquid mustard gas has been placed on the skin the time interval between contamination and successful decontamination is greatly decreased.

(c) As the only practical way of preventing skin burns from high concentrations of vapour is the wearing of chemically impregnated clothing, there results a very difficult problem. The use of impregnated clothing presupposes that the body surface is fully covered, which in the tropics is a difficult requirement to meet. In addition the washing of impregnated clothes removes their protective value and since in the tropics frequent washing of clothes is desirable on hygienic grounds, the maintenance of adequately protected clothing would be difficult.

Notes on Decontamination

109. Satisfactory decontamination requires care and attention but demands no high degree of specialized knowledge and can therefore be carried out by all trained troops.

(a) Stretchers

The ordinary pattern is difficult to decontaminate completely since the canvas is fixed to the woodwork by nails, rendering its removal and replacement impracticable under field conditions. It is therefore very important to prevent contamination occurring. Stretchers should, therefore, when not in use be kept indoors or under cover to prevent splashing with liquid mustard gas. As an additional precaution, they should also be protected by an anti-gas stretcher cover of special laminated paper of the type issued during World War II which will prevent or delay contamination of the canvas when used for contaminated casualties. If the standard cover is not available protection may be improvised with a ground sheet or anti-gas cape, which can be decontaminated after use by one of the standard methods. Special oilskin sleeves were available in World War II to protect the handles of the stretcher.

If contamination does occur the complete stretcher should be scrubbed with bleach paste and then washed thoroughly with water, after which it should be left to weather as long as possible. This procedure does, however, damage the canvas. The handles may not be safe for the bare hands for several days and, therefore, anti-gas gloves should be worn. Anti-gas ointment, well rubbed into the hands, may be used prophylactically when handling contaminated material or casualties. Any danger from residual contamination on the canvas can be lessened by using the covers described above.

If the canvas can easily be removed it can be decontaminated by immersing it in boiling water for 30 minutes.

Stretchers which have been exposed to mustard gas vapour only should be allowed to weather in the open air.

(b) Clothing and Equipment

Articles exposed to mustard gas vapour only, should be hung in the open air for 6–12 hours.

(i) *Cotton and woollen* articles should be boiled in water for one hour. The same water should not be used more than three times.

(ii) *Boots*—these can be rendered free from contamination by immersion in hot water (120–130°.F.) for 2 hours followed by drying, preferably in a warm atmosphere, but this makes them no longer serviceable.

(iii) *Capes and oiled fabrics*—these should be immersed in water at 200-210°.F. for half an hour. After this treatment they should be hung up, preferably in a warm atmosphere.

(iv) *Rubber articles*—these should be boiled for the following periods:—

    (1) Ground sheets and similar articles of thin rubber .. 1 hour

    (2) Rubber gloves, boots, etc. .. 2 hours

They should afterwards be dried in a warm atmosphere.

(v) *Metal articles in general*—weapons, instruments and similar articles should be swabbed with gasoline or kerosene or may be boiled for 60 minutes. Bleach paste is also satisfactory but as it corrodes metals it must be removed from metal objects within 10 minutes of application.

THE NITROGEN MUSTARDS

110. These are oily, colourless or pale yellow liquids, soluble in organic solvents but only slightly soluble in water. The products of their hydrolysis are toxic. Some of the nitrogen mustards have a faint fishy smell, others have no smell.

111. The symptoms produced by these substances are similar to those produced by other blister gases—mild exposure producing irritation and lacrimation of the eyes within about 20 to 30 minutes, but skin symptoms may not occur. After severe exposure there is erythema of the skin and mild but fairly rapid blister formation. The respiratory tract is affected in a manner similar to that following mustard gas inhalation.

The most marked effects of the nitrogen mustards are on lymphatic and haemopoietic tissues and these effects may follow absorption from the skin and the respiratory tract. The actions on the bone marrow are very marked and produce effects ranging from a mild transient leucocytosis to severe leucopenia, thrombocytopenia and anaemia.

112. Treatment of poisoning by nitrogen mustards is the same as that for mustard gas.

ARSENICAL SKIN IRRITANTS (or ARSENICAL BLISTER GASES)

Introduction

113. Substances included in this group are Lewisite and the Dicks. Their blistering action is essentially the same as that of mustard gas but their systemic effects differ in that they are complicated by arsenical poisoning. The hazard from these substances has been greatly reduced by the development of British Anti-Lewisite (BAL). This was discovered after considerable research during World War II in an attempt to devise an antidote to the arsenical vesicant gases.

Lewisite

114. *Lewisite*, or chlorovinyl dichlorarsine, is an important chemical compound developed towards the end of World War I. It has never been used in war and consequently knowledge of its action has been acquired by laboratory experience and not by a study of military casualties.

In the pure state Lewisite is a powerful toxic substance embodying the aggressive qualities of the lung irritant gases, the irritant characteristics of the tear and nasal gases, and the vesicant properties of the " blister " gases. Under modern conditions of chemical warfare, however, its vesicant action would predominate; hence its inclusion in this group.

Chemical and Physical Properties

115. Lewisite is a vesicant liquid which freezes at about −13°C. (8·6°F.) and is soluble in organic solvents, oils and fats. Water rapidly causes hydrolysis, especially in the presence of alkalis. The products of hydrolysis are also highly toxic (though not volatile) and if absorbed in sufficient quantity from the skin may cause acute arsenical poisoning. It is also hydrolysed on soils, especially alkaline soils.

The impure material has a geranium-like smell and the vapour has an immediate and strongly irritant effect on the nose and throat. Lewisite vapour does not penetrate the clothing; the liquid, however, penetrates clothing, rubber and oilskin more quickly than liquid mustard gas, but wet clothing gives fair protection.

Clinical Effects

116. Symptoms of shock accompanied by haemoconcentration may result from a heavy contamination of the skin with liquid lewisite. The liquid also causes severe burns of both the skin and the eyes. It penetrates the tissues

rapidly and in the skin produces an unpleasant stinging sensation with irritation and blistering. Lewisite vapour is strongly irritant to the eyes and the respiratory tract, but on the skin it usually causes no more than erythema. Erythema due to liquid lewisite develops more rapidly than that of liquid mustard gas and vesication, when it occurs, is usually fully developed within 6 to 8 hours. Unlike the mustard gas blister, the lewisite blister is sharply defined, covers most of the erythematous area and is filled with an opaque or opalescent fluid.

117. *Effects on the eyes.*—Liquid lewisite causes immediate pain, spasm of the lids and lacrimation. Within 15 minutes, there is intense chemosis, sub-conjunctival haemorrhage and oedema of the cornea. Within 3 to 4 hours, the lids adhere to their margins, the cornea is hazy, and the conjunctiva is extensively ulcerated. Severe complications such as corneal ulceration and sloughing, or iritis and hypopyon may follow.

Dicks

118. The Dicks are arsenical liquids, the best known of which is ethyl-dichlorar-sine—a clear, oily liquid with a fruity smell. These gases are not as persistent as the other " blister " gases. They persist only for 2 to 3 hours under normal European weather conditions and are unlikely to be used in a future war.

They are intensely irritating to the nose, thus possessing harassing properties similar to those of the nose gases. The vapour, if inhaled, produces effects similar to the lung irritants. The action of the liquid and gas on the eyes is similar to that of lewisite and on the skin produces erythema and blistering.

Treatment of Lewisite and Dick Casualties

119. *British Anti-Lewisite (BAL)* is 2:3-dimercaptopropanol, $CH_2H.CHS$ $SH.CH_2OH$. It is a colourless, mobile liquid with an onion-like smell and is approximately 6 per cent soluble in water at room temperature.

It combines with compounds of arsenic (and of mercury and other heavy metals) to form a five-membered ring compound which is more stable than that formed between the arsenical and the tissue-acceptor. The resulting compound is rapidly excreted by the kidneys. The blistering effects of lewisite on human skin can be prevented by the local application of BAL to the affected area as late as $1\frac{1}{2}$ hours after contamination. Its application can therefore be delayed until after intense erythema has appeared, affording a practical indication to the effect that BAL can *reverse* as well as protect against the toxic action of arsenicals. The results of animal experiments have shown that in the case of contamination of the eyes the instillation into the conjunctival sac of 5 to 10 per cent solution of BAL within 5 minutes of contamination resulted in almost complete recovery.

For inunction into contaminated skin 5 or 10 per cent ointments have been employed. For the treatment of systemic vesication 5 or 10 per cent BAL in arachis oil and benzyl benzoate may be given by deep intramuscular injection.

Details of Treatment

120. When liquid lewisite has entered the *eye* speed in treatment is essential. BAL ointment should be rubbed along the lid margins: this will reduce the pain. The lower lid can then be pulled down and more ointment inserted into the lower conjunctival fornix. BAL ointment may be massaged gently into the eye through the closed lids.

BAL ointment may be used successfully in the treatment of skin injuries. The ointment is itself an irritant and can cause erythema of the skin and acute discomfort of the eye, but these effects are transient. However, inunction with B.A.L. ointment may still be successful at the erythematous stage.

Intramuscular BAL should be given to counteract the toxic effects of systemic absorption. An intravenous preparation of BAL has been prepared and may be used if available.

The shock associated with poisoning by the arsenical " blisters " should be treated by standard methods.

121-130. *Reserved.*

CHAPTER V

LUNG IRRITANT OR CHOKING GASES

Introduction

131. These gases exert their main action on the lungs and respiratory passages, causing pulmonary oedema after a latent period. Phosgene is the most important gas of the group and in World War I proved to be the most effective. The action of phosgene on the respiratory tract may be regarded as typical of that exerted by other members of this group. (Chlorine however in high concentrations causes instantaneous symptoms). (*See* Chlorine—Chapter XI).

Phosgene

Chemical and Physical Properties

132. Phosgene (Carbonyl Chloride), $COCl_2$, is a colourless, volatile liquid which boils at 8·2°C. (46·8°F.) with the evolution of a colourless gas. It has a highly characteristic odour of musty hay, even in harmless concentrations.

Methods of Dispersion

133. Phosgene may be dispersed by shell, both air and ground burst, and by cylinder. Since at normal temperatures it is a non-persistent gas, its successful use would depend on the prevailing weather conditions. Phosgene might also be used with other gases, *e.g.*, mustard gas.

Detection

134. (*a*) *Odour.* The gas may be readily detected by its characteristic odour, even in low concentrations, but the sense of smell tires very quickly. In high concentrations such as a heavy cloud in an enclosed area or in close proximity to a source, the gas constitutes a greater hazard since an intense bronchospasm may be induced by a single breath, interfering with the rapid adjustment of the respirator.

(*b*) *Chemical.* Chemical indicators change colour in the presence of phosgene.

Mode of Action

135. (*a*) *Respiratory system.* The gas may cause some bronchiolar constriction as it is inhaled. The permeability of the alveolar capillaries is increased and oedema fluid gradually fills the alveoli. The oedema fluid interferes with the gaseous exchange of both oxygen and carbon dioxide and this calls for increased respiratory effort, resulting in localized emphysematous areas. On account of these pathological changes, oxygen want becomes increasingly urgent and results in impaired heart function.

(b) Circulatory system. The loss of fluid from the circulation into the alveoli produces a marked haemo-concentration, and severe cases may show haemoglobin percentages as high as 140, with corresponding increase in the red cell count. The resultant increase in blood viscosity, together with the oxygen want from impaired respiratory exchange, causes embarrassment to the heart. This aggravates still further the anoxia so that a vicious circle is established, resulting ultimately in cardiac failure.

Pathology

136. The essential lesions are pulmonary oedema, rupture of the pulmonary alveoli, and concentration of the blood, with increased viscosity and a tendency to thrombosis.

Frothy fluid may be seen escaping from the nostrils and mouth. On opening the chest the lungs are seen to be voluminous, oedematous and congested. The sooner death has occurred after gassing, the greater the degree of pulmonary oedema, but intense pulmonary oedema may be found in cases who have died after a long latent period.

The trachea and bronchi after phosgene poisoning are usually normal in appearance, but contain much frothy yellowish fluid. This contrasts with chlorine and chloropicrin poisoning, however, in which the bronchi and even the trachea may show serious damage; the epithelial lining may be severely affected and desquamation may take place. The small bronchi may appear mildly inflamed.

On removal from the chest the lungs are heavy with oedema, but it is quite unusual to find them uniformly water-logged. Areas of greatly oedematous lung alternate with areas in which there is emphysema and distension, together with small depressed areas of collapse. When the lung is sectioned, serous fluid drips from the cut surface and very dark viscid blood exudes from the cut vessels. Free fluid in varying amount is usually found in the pleural cavity.

The right side of the heart may show dilation but, apart from venous congestion, the other organs in the body are generally normal both to the naked eye and on histological examination.

Signs and Symptoms

137. Exposure to an atmosphere containing phosgene causes immediate sensory irritation of the respiratory passages, accompanied by smarting and watering of the eyes. This irritation of the respiratory passages causes catching of the breath, coughing and a sensation of tightness and constriction and pain in the chest. After the initial check, the breathing continues, but is gasping in character and interrupted by violent fits of coughing. After the patient gets out of the poisonous atmosphere the respiration remains rapid and shallow, any attempt to draw a deep breath giving rise to painful discomfort and provoking a fit of coughing. Nausea, retching, and vomiting are prominent features in the early stages of poisoning and may make it difficult to wear a respirator. There is slight or profuse expectoration. Headache, and a sense of fatigue in all the limbs, often prostrate the patient.

It is important to note that the severity or otherwise of the initial symptoms are unreliable in predicting whether any subsequent pulmonary oedema will be mild or fatal.

There usually follows a latent period of perhaps several hours' duration, during which little discomfort is felt, except possibly some breathlessness on exercise. Severe exertion may precipitate serious or even fatal respiratory or cardiac symptoms. When gassing has been severe the latent period is short or almost absent and serious symptoms develop early.

After the latent period breathing becomes rapid, shallow and difficult. The cough may return, with the expectoration of much frothy white or yellowish fluid, several pints of which may be coughed up in a few hours. Haemoconcentration begins early and may be considerable.

As pulmonary oedema increases, a condition of " internal asphyxia " with acute oxygen lack is apparent. This causes cyanosis which is due (a) to deoxygenation of the blood and, (b) to relatively increased corpuscular content which results from haemoconcentration. In the early stages there is a florid generalized type of cyanosis. Later, in the stage of circulatory collapse, there is a leaden pallor which may be detectable only in the lips and ears because of the generalized pallor. There is a weak running pulse (130 to 140 per minute) and respiration is rapid and shallow. Some cases exhibit restlessness and anxiety while others fall into a semi-coma with a muttering delirium. These stages may be quickly followed by collapse and death. Of the fatal cases, 80 per cent die in the first twenty-four hours, and very few die after the third day. When deaths occur later than this they are usually due to bronchopneumonia.

Many cases do not pass on to the pallid state, but remain in the state of florid cyanosis of the face and neck, possibly due to continuing haemoconcentration. Most of these cases recover. As a rule, recovery is rapid after the third day. The absorption of the oedema fluid in the lungs takes place quickly and by the end of a week the patient is semi-convalescent. The pulse rate is often slowed to about 50 per minute or even less as the acute stage subsides. This transient bradycardia may be seen earlier in mild cases.

On examination of the chest, the percussion note may remain resonant, notwithstanding the existence of pulmonary oedema. The breath sounds are weakened, especially over the back; they may be harsh in character but never tubular. Fine râles are heard, chiefly in the axillary region and at the back and sides of the chest, while rhonchi may be noted occasionally.

In the early acute stage the chest signs give little indication of the gravity of the case or the extent of the damage to the lungs. The early diagnosis of pulmonary oedema is possible by means of X-ray examination. The colour, the pulse and the character of the respiration are the chief guides to prognosis. With the development of inflammatory complications and rising temperature the physical signs become those of pleurisy, bronchitis and broncho-pneumonia.

Once the patient is out of danger the convalescence may still be prolonged by a wide variety of troublesome symptoms such as lassitude, dyspepsia, precordial pain, dyspnoea, and an exhausting and persistent tachycardia even after mild exercise.

Recurring attacks of spasmodic dyspnoea may occur during the night and cause the patient considerable alarm. Such cases usually have a definite polycythaemia.

Prognosis

138. In the early stages, prognosis should be guarded because of the insidious nature of the poisoning. The majority of deaths occur within the first twenty-four hours, and those that survive longer than three days usually recover. Cases exhibiting florid cyanosis in the acute phase generally have a good prognosis, whereas the cases which develop a grey leaden appearance frequently die of cardiac failure. Casualties which survive the acute phase may ultimately succumb to broncho-pneumonia which may develop later, although this hazard may be reduced by the use of antibiotics.

Protection

139. The respirator affords complete protection and unless personnel are suddenly exposed to a high concentration, the smell of the gas will cause them to put on their respirators quickly.

Treatment

140. *First Aid Treatment.* As already stated it is seldom possible to forecast the severity of the subsequent illness, if any, by a clinical examination soon after gassing. The early symptoms are largely subjective, such as tightness in the chest. There may be vomiting and coughing, but in the chest no physical signs may be detectable. In this early stage after gassing, military personnel should not be regarded as more than potential casualties and should normally remain at their post of duty. In World War I it was common to regard such subjects as casualties and many were sent to hospital unnecessarily, frequently as stretcher cases.

At that time there was a definite impression that any exercise during the latent period after inhaling the gas increased the severity of any pulmonary oedema which might follow later, and for this reason strict rest was imposed whenever possible from the time of gassing. Experimental work on animals carried out since, however, has not supported this view. None the less, severe exercise should be avoided when possible after even mild phosgene poisoning. It is difficult to outline precisely what action a medical officer should advise after a wave of phosgene gas has passed over unprotected personnel. To have them sent to hospital immediately is out of the question. On the other hand, to classify everyone as fit might result in a minority of the " potential " casualties becoming rather worse as a result.

The action of the medical officer should depend on two factors:—

(*a*) In an urgent military situation it would not be expedient to consider as a casualty any man who is reasonably capable of carrying out his duties. If, however, symptoms recur at the end of the latent period, he should be considered a casualty and treated as such, any undue exertion being avoided.

(*b*) In a less urgent situation it is still desirable that potential casualties should carry on until definite objective symptoms declare themselves.

141. *Hospital Treatment.* There are three absolutely essential requirements in the treatment of a gassed patient who shows signs of respiratory distress due to pulmonary oedema. They are: strict rest in bed, warmth and oxygen.

(*a*) *Rest* may be disturbed by anxiety, restlessness and coughing. Should restlessness be severe it may be desirable to give codeine. If the patient is apprehensive the physician should weigh the merits of sedation against the undesirability of further depressing the respiratory centre.

(*b*) *Warmth*, but not over-heating, is necessary to combat shock.

(*c*) *Oxygen.* The value of oxygen cannot be over-emphasized. It should be given early, freely and continuously by the best means available. Six to eight litres per minute should be given and it should be inhaled direct (after warming if possible) and not merely be used to enrich the inspired air. This not only increases the oxygen saturation of the blood as shown objectively by the improved colour of the patient, but is the best form of supportive treatment for the heart in distress. The increased feeling of well-being in the patient while breathing oxygen goes far to allay the restlessness and anxiety of these anoxaemic patients. Drainage by raising the feet and lowering the head for a few minutes, if not too distressing, was found useful in World War I.

142. *Venesection.* Although animal experiments have provided no support for this treatment, it was reported in World War I that venesection had a definite

place in the therapy of those cases who were cyanosed and had dilatation of the right side of the heart, but it was considered to be definitely harmful in the pale, severe, collapsed cases. Its real place in treatment remains doubtful, and it is therefore *not* recommended.

143. *Other Treatment.* So far as the value of drugs and other therapeutic measures is concerned, the following depressing lists record treatments which have been tried, mostly in experimental animals, and found to be either *(a) of no real value*, or *(b) positively harmful*.

    (a) Of no real value:—
> (i) Antispasmodics—may temporarily relieve difficult breathing but are liable to cause undesirable tachycardia.
> (ii) Expectorants.
> (iii) Raising the serum calcium by injection of calcium lactate or gluconate, or with parathormone.
> (iv) Cardiac stimulants in general.
> (v) Ascorbic acid therapy.
> (vi) Adrenocortical hormone therapy.
> (vii) Dry air for 24 hours after gassing.

    (b) Positively harmful:—
> (i) Transfusion with serum, plasma or whole blood, whether normal or concentrated, also transfusion with saline or 50 per cent glucose. Oedema of the lung is thereby increased.
> (ii) Intravenous or subcutaneous administration of oxygen.
> (iii) Cooling the body by sponging or refrigeration.
> (iv) Atropine—does nothing to limit pulmonary oedema. Large doses produce undesirable cardiac acceleration.
> (v) Diuretics.
> (vi) Intravenous Heparin or Dicoumarin.
> (vii) The use of carbon dioxide-oxygen mixture.
> (viii) Artificial pneumothorax, alone or followed by a positive intra-thoracic pressure.

Complications

144. A few cases may develop broncho-pneumonia as the oedema subsides. Penicillin or the sulphonamides are then indicated. The usual precautions during sulphonamide therapy must be observed.

Convalescence

145. Return to normal activity after the period of absolute bed rest during the acute stage must in every case be gradual. Full duty should never be allowed until the response to gradually increased exercise has been assessed by the physician. It is not unusual to find that although a man looks and feels well while at rest, he quickly gets exhausted, breathless, and has a rapid pulse even after mild exercise.

    The cases which suffer from attacks of nocturnal asthma with polycythaemia are more difficult to cure, but the effort should be made to prevent such men regarding themselves as chronic invalids.

Sequelae

146. There is no conclusive evidence either from clinical, radiological or autopsy examination, that pulmonary fibrosis, chronic bronchitis or emphysema follow exposure to lung irritant gases. Nor is there any evidence that the incidence of tuberculosis is higher in soldiers who have been exposed to the gases. (*See* Price, G. Basil, Med. Press and Circular, 16th March, 1938).

147-150. *Reserved.*

SECTION II—WAR GASES OF LESSER IMPORTANCE

CHAPTER VI

LACRIMATORY OR TEAR GASES

General Description

151. This group includes certain gases or irritant smokes which, as met with in the open field, cause symptoms of distress without any subsequent physical injury. Typical examples are the following:—

Lacrimators or " tear gases," such as:—

Ethyliodoacetate (K.S.K.).
Bromobenzyl cyanide (B.B.C.), and
Chloracetophenone (C.A.P.).

The primary objects in the employment of these compounds is for use as anti-riot weapons or to harass troops by causing an acute, though temporary, distress, thereby compelling them to wear respirators, possibly for prolonged periods. This tends to reduce efficiency, and, if anti-gas training be defective, to have a lowering effect on morale.

These gases have several important features in common, *viz:*—

(a) Their action is selective, *i.e.*, they only attack exposed sensory nerve endings in mucous membranes, such as those of the eye, the naso-pharynx and the respiratory tract.

(b) The effects are immediate, but transient. Withdrawal from the hostile atmosphere is followed by recovery.

(c) They are effective in extremely low concentrations, but they never give rise to permanent disabilities. It is unusual to incur any physical injury from these irritating gases.

Chemical and Physical Properties

152. There are many compounds, both liquid and solid, which may be used as lacrimators in wartime. The following are typical examples:—

(a) Ethyliodoacetate.—A dark brown, oily liquid with a smell resembling that of " pear drops." Its high boiling point (180° C. or 356° F.) and comparatively low vapour pressure ensure for it a certain degree of persistence on the ground.

(b) Bromobenzyl cyanide.—In the pure state this is a yellowish crystalline solid, stable at ordinary temperatures and melting at 24·8° C. (74·6° F.). In the crude form, as employed in war, B.B.C. is a heavy, oily, yellow liquid with a penetrating, bitter-sweet smell. The liquid boils at 242° C. (467·6° F.); it is more stable, and has a lower vapour pressure than ethyliodoacetate, hence it persists longer than the latter as an effective lacrimator when spread on the ground.

(c) Chloracetophenone.—A colourless, crystalline solid melting at 54° to 59° C. (129·2 to 138·2° F.) and boiling at 245° C. (473 °F.). Though only sparingly soluble in water, it dissolves readily in all the organic solvents. Chloracetophenone is a very stable compound which does not decompose on heating or detonation; its lacrimatory effects, however, are soon lost by reason of its condensation to the solid, inert state soon after the initial dispersion, and it is therefore classed among the non-persistent gases. It is used extensively in gas training schools.

Methods of Dispersion

153. The liquid lacrimators may be dispersed by shell or bomb, or by mechanical spraying for purposes of ground contamination. The solid C.A.P. is dispersed from generators by means of heat, or it may be sprayed in solution, from the exhaust pipes of internal combustion engines. It is, however, unsuitable for use in shell.

For training purposes, small capsules containing solid C.A.P. are heated over a flame to evolve the gas. In certain countries effective use of this compound is made by the police against mobs; a variety of more or less harmless weapons such as lacrimatory pistols and hand bombs are employed for this purpose.

Mode of Action

154. Exposure to any of these lacrimators gives rise to an immediate, acute and localized irritation of the sensory nerve-endings on the corneal and conjunctival surfaces, which may vary from a mild irritation to an intense stinging sensation according to the concentration. Through reflex action, this is followed by profuse watering of the eyes and spasm of the eyelids, and the latter may be so acute as to render it impossible to keep the eyes open.

With a rise in the concentration of the vapour, further effects may make their appearance. The irritant action of the gas on the respiratory passages and lungs produces a burning feeling in the throat and discomfort in the chest, and, if the exposure be continued, nausea and vomiting may result.

Ethyliodoacetate is less irritating to the respiratory tract than chloracetophenone. The latter, however, unlike the liquid lacrimators, will irritate the bare skin, especially if it be hot and moist.

As a rule, persons exposed to lacrimators do not exhibit more grave symptoms than those described above, as the very high concentrations necessary to produce lung lesions are not met with in the field. In confined spaces, however, where the actual liquid may be splashed on the skin, or the gas inhaled in high concentration, worse results may be expected. These vary from severe conjunctivitis, with tracheitis and bronchitis from the effects of the vapour, to blistering of the skin, keratitis and corneal opacities after contamination with the liquid.

Protection

155. The respirator affords complete protection to both eyes and lungs against all concentrations of tear gases likely to be met. The use of goggles alone is not recommended, as, in addition to the liability of leakage and constant dimming, they offer no protection to the respiratory tract.

The irritant action of C.A.P. and other solid lacrimators on the hot, moist skin is not sufficiently intense in temperate climates to necessitate the use of special protective garments.

Treatment

156. In the great majority of cases, adjustment of the respirator will suffice to alleviate the symptoms, and, usually, to clear up the condition completely. Experience has shown that even after severe exposures all symptoms disappear within 12 hours. No treatment is likely to be necessary, and there should be no after-effects. In the rare cases where conjunctivitis or respiratory affections develop, treatment should be symptomatic and follows general principles. If the eyes are contaminated by the actual liquid from aerial sprays, bursting bombs, etc., treatment should follow the lines laid down for mustard gas (Chapter IV).

157-160. *Reserved.*

CHAPTER VII

NASAL IRRITANTS OR NOSE GASES

General Description

161. These sensory irritants are solid organic arsenical compounds which can be dispersed by heat or detonation in the form of a very fine, almost invisible, particulate cloud or smoke.

They were employed in World War I in the belief that they would penetrate the respirator then in use and cause such distressing symptoms that the men would discard their respirators as useless; this would expose them to the effects of lethal gases, such as phosgene, which were often released simultaneously. The nasal irritants did not, however, meet with any striking success, because at that time the methods of release were unsatisfactory.

Chemical and Physical Properties

162. The following are typical examples of the nasal irritants:—

(a) Diphenylamine-chlorarsine (D.M.).—A yellow, almost odourless, crystalline solid melting at 196° C. (383° F.) and boiling (with decomposition of the compound) at 410° C. (770° F.) at ordinary atmospheric pressure; insoluble in water, and difficult to dissolve in the ordinary organic solvents.

(b) Diphenylcyanarsine.—A colourless crystalline solid, almost entirely odourless, with a melting point of 33° C. (91·4° F.) and boiling at 346° C. (654·8° F.) at ordinary atmospheric pressure; almost insoluble in water, but dissolving readily in oils and in organic solvents.

It may be noted that diphenylchlorarsine (D.A.), another member of the group, and diphenylcyanarsine have slight vesicant properties. They are chemically related to chlorovinyldichlorarsine (lewisite) and ethyldichlorarsine, which are also sensory irritants though their vesicant action is of greater practical importance.

Methods of Dispersion

163. These compounds are solids at ordinary temperatures, but when heated or detonated they are vaporized, without decomposition, in the form of an almost invisible cloud of minute particles which remain suspended in the air.

They may be heated by means of special incendiary generators which are placed in position by hand or dropped from vehicles or aircraft, or the solid compound may be incorporated in shell. By these methods it is possible to emit, over a wide front, a cloud of extremely fine particles.

Mode of Action

164. The main feature of these arsenical irritants is their power of causing violent sensory irritation in man even though present in extremely low concentrations.

Their effectiveness depends on the amount inhaled during the few minutes before symptoms come on. Even if the affected person withdraws from the poisonous atmosphere (or puts on his respirator) directly the irritant effects are felt, the symptoms continue to increase in severity for some time before they begin to subside. Whereas the tear gases cause immediate irritation which diminishes as soon as the eyes are protected, the arsenicals have a delayed action which may cause its maximum of distress a little while after the respirator has been adjusted. Unless personnel are trained to expect this, they may lose faith in the respirator, with disastrous results.

The symptoms are characteristic, and consist in acute pain in the nose and accessory sinuses, with a sense of " fullness " in the head and with repeated

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sneezing (hence the term " sternutators " applied to these gases). A burning sensation in the throat, and one of tightness and pain in the chest, also a feeling of grittiness in the eyes with pain and lacrimation, and aching of the gums are common, while salivation, nausea and even vomiting, are important. Mental distress is very marked in severe cases, in which the patients feel and look utterly miserable. This condition of intense discomfort is very alarming to the inexperienced.

The effects, however, are transitory, and affected men should not leave their units as the symptoms usually disappear within one to four hours. During World War I cases were reported showing paralyses of limbs, which suggested a central toxic action of the gas; but recovery took place within 24 hours. Various transient paræsthesiæ developing later were regarded as probably functional, and no lasting organic lesion is likely to follow exposure to these gases in the open.

Protection

165. The respirator charcoal, so effective in arresting the lethal gases, has little protective value against the particulate clouds of these arsenical compounds. Special filtering devices must be employed to trap the arsenical dust in the air, and all modern respirators for use against war gases give adequate protection.

Symptoms of true arsenical poisoning may occur through men using water drawn from shell craters contaminated with these arsenicals. The arsenic content in these craters may be very high, and men have been known to suffer from dermatitis after shaving with water drawn from them. It is important, therefore, that stringent orders be issued that no water from shell craters be employed for drinking, cooking or washing.

Treatment

166. In the great majority of cases a brief period of rest is the most that is required. In a few exceptionally severe cases, however, pain may call for relief from the medical officer, when the inhalation of a little chloroform will be found useful. A five per cent. solution of sodium bicarbonate for nasal irrigation or as a gargle will help to allay the irritation of the nose and throat. Benzedrine (amphetamine) has been recommended for depression.

If severe poisoning should occur, *e.g.*, after drinking contaminated water, BAL may be given intramuscularly to assist in the elimination of arsenic.

167-170. *Reserved.*

CHAPTER VIII

PARALYSANT GASES

Hydrocyanic Acid (Prussic Acid)

History

171. Hydrocyanic acid has long been recognized as one of the most powerful poisons known to man. It was used in World War I as a shell filling, but proved ineffective because the gas dispersed too rapidly. The relatively low concentrations which resulted were not lethal. Nevertheless, study of this gas is essential as it may be used in the future with greater success under different conditions.

Chemical and Physical Properties

172. Hydrocyanic acid is a clear, colourless liquid of low boiling point (26° C. or 78·8° F.), very volatile and smelling strongly of bitter almonds. (Some persons, as an idiosyncrasy, are unable to distinguish the smell of hydrocyanic acid). It is very soluble in water and in alcohol. Watery solutions do not redden litmus paper.

The vapour of hydrocyanic acid is somewhat lighter than air and diffuses rapidly when released. In closed spaces it is extremely toxic; in the open, however, the dispersion of the gas is so rapid that relatively low concentrations, which are not lethal, result. This explains the failure of hydrocyanic acid gas shells in World War 1 in the open field, where they caused but few casualties.

Mode of Action

173. The gas arrests the activity of all forms of living matter by inhibiting oxidation.

In high concentration, such as may be found in a confined space, this gas may well be considered a fulminant poison, as it may cause death with dramatic rapidity through paralysis of the respiratory centre in the brain. In low concentration it may be detoxicated in the body as quickly as it is absorbed.

Attention may be drawn to a danger in the use of hydrocyanic acid gas when it is employed for the destruction of vermin, such as rats in ships, or when used in the disinfection of rooms. A suitable respirator eliminates the danger of its inhalation, but since this gas may be absorbed by the skin it is dangerous to remain long, even with a respirator, in the high concentrations employed against vermin.

Liquid HCN can also be absorbed through the skin and from the eyes in amounts sufficient to produce death. This risk is accentuated if evaporation from the skin is prevented by overlying oilskin clothing.

Owing to the ease with which the gas dissolves in water, the skin absorption danger is greatly increased if the weather be hot and the skin bathed in sweat.

Symptoms

174. The gas is not irritant to eyes, nose or throat. With high concentrations the effects are rapid. The symptoms are ushered in by uneasiness and vertigo, palpitation and deep hurried breathing; unconsciousness and convulsions follow quickly, and death occurs through paralysis of the respiratory centre and failure of the circulation.

Concentrations that are not lethal may produce headache or giddiness, and sometimes nausea or inability to concentrate; recovery, however, is usually rapid and complete.

Treatment

175. The aim in treatment is to keep the victim alive until detoxication mechanisms permit resumption of activity by the poisoned enzyme systems. The patient must be removed from the dangerous atmosphere. If the intoxication it due to liquid absorption through the skin then the contaminated clothing should be removed. Oxygen should be given if there has been prolonged apnoea. Warmth and rest are essential. Amyl nitrite inhalations should also be used. If breathing has stopped, artificial respiration should be administered.

Laboratory experiments have shown that cobalt salts are strongly antidotal to cyanide. The suggested dose for an adult is 1·5 gm. of cobalt acetate in 20 ml.

of water by intravenous injection. This treatment is a drastic measure and should only be tried if poisoning is very severe. Cobalt salts produce strong purgation. The inhalation of amyl nitrite and the intravenous injection of 10 ml. of 3 per cent sodium nitrite, followed by 50 ml. of 25 per cent sodium thiosulphate, has also been recommended for the treatment of cyanide poisoning.

The possibility of permanent mental damage after prolonged unconsciousness from cyanide poisoning should be borne in mind.

Cyanogen Chloride

Chemical and Physical Properties

176. Cyanogen chloride is a volatile " non-persistent " colourless liquid. Although only slightly soluble in water it dissolves readily in organic solvents. It boils at 15·8° C., freezes at −7° C., and gives off an irritant vapour which in low (non-toxic) concentrations quickly causes stinging in the nose, smarting of the eyes, severe lacrimation and blepharospasm. In contrast to hydrocyanic acid, therefore, good warning of risk is given, and in fact the immediate reaction of persons exposed is to hold the breath. The vapour is twice as heavy as air. In closed spaces, toxic concentrations may be encountered.

Mode of Action

177. The properly fitting respirator gives complete protection since oral or skin absorption is not likely. Cyanogen chloride on absorption from the lung alveoli reacts with the haemoglobin of the red corpuscles and forms a compound from which hydrocyanic acid is liberated. Besides the irritant effects to the eyes and throat, therefore, inhalation of larger quantities produces a group of systemic symptoms similar to those caused by hydrocyanic acid. The first effects are dizziness, headache and dyspnoea. Respirations become rapid and shallow and unconsciousness quickly follows. Convulsions may occur. Death is due to general inhibition of the oxidative enzymes involved in tissue respiration, and central respiratory failure. In non-fatal cases pulmonary oedema, which may begin in a few hours, is a common complication.

Protection

178. Satisfactory protection is *only* obtained with a respirator containing charcoal which has been specially impregnated with pyridine or other suitable substances. The present types of civilian and service respirators give adequate protection against cyanogen chloride. Even with fully impregnated charcoals, cyanogen chloride is the least readily absorbed war gas, and consequently the standard of protection provided affords an additional margin of safety against other agents—including hydrocyanic acid and arsine.

The onset of irritation or lacrimation when wearing the respirator is a warning that the charcoal is becoming saturated.

Treatment

179. If the amount of cyanogen chloride is sufficient to cause systemic symptoms, urgent measures similar to those required for hydrocyanic acid poisoning are needed. The patient must be removed at once from the poisonous atmosphere. Clothing must be loosened and oxygen should be given. Warmth and rest are essential. Amyl nitrite inhalations should be used. If breathing has stopped artificial respiration should be administered, with oxygen.

180-185. *Reserved.*

SECTION III—OTHER DANGEROUS GASES

CHAPTER IX

ARSINE

Chemical and Physical Properties

186. This is a colourless, inflammable gas which, when liquefied, boils at −55° C. It is formed, together with hydrogen, when acids react with metals containing arsenic as an impurity, and is readily evolved by the action of water on calcium, magnesium and sodium arsenides, or of dilute acid on other metallic arsenides.

It may be used in chemical warfare as a gas in cylinder attacks, or as a non-persistent charging in shells and bombs. In addition, one of its progenitors, *e.g.*, calcium arsenide, might be disseminated in the form of a dark-grey heavy powder, which in contact with atmospheric and soil moisture would slowly generate arsine *in situ*, over a period of hours or days according to conditions.

In moderately strong concentrations, arsine possesses a nauseating garlic-like odour and gives rise to a metallic taste in the mouth; but when largely diluted with air, it may be imperceptible to the senses although possibly present in toxic concentrations. It is non-irritant to the eyes, the skin and the respiratory passages and is rapidly absorbed through the lungs into the body.

Exposure for a few minutes to a high concentration of arsine may be rapidly fatal. Even extremely low concentrations, however, can cause symptoms of poisoning, though slow in onset and mild in degree, provided that the exposure is long enough, *i.e.*, many hours or even days. Work in a confined space should, therefore, not be permitted where there is any risk of arsine poisoning, especially as arsine exhibits a cumulative effect in the body.

Pathology

187. Arsine is taken up chiefly by the red blood corpuscles, the most characteristic feature of its action being to cause acute haemolysis, with consequent anaemia. The liberated haemoglobin, some of which becomes altered to haematin and possibly to methaemoglobin, is partly excreted by the kidneys, giving rise to haemoglobinuria, and partly converted by the liver into bile pigments. The ' bronzed ' jaundice is due mainly to this haematin, and also partly to bile pigments because of the above mechanism, as well as liver damage due to anoxaemia. The haemolysis may continue for several days after exposure, and the anaemia is therefore progressive. While haemolysis is taking place a simple estimation of the haemoglobin percentage in the whole blood will give an erroneous idea of the extent of the cell destruction, since much of the haemoglobin may be in solution in the plasma and not in the corpuscles. A red cell count will clarify the situation, though in most of the intact red cells some haemoglobin has been inactivated by the arsine.

Even in the early stages of poisoning there is evidence of increasing damage to the kidneys and liver, and these organs are intensely congested. This is soon followed by tubular degeneration in the kidneys, with the excretion of blood casts, and toxic degeneration and necrosis of the liver. In the most severe cases complete anuria occurs soon after exposure.

Death may occur within 48 hours from acute systemic poisoning by arsenic. Pulmonary oedema and cerebral oedema have been described in some of these cases. At one time it was suggested that the anuria was caused by blockage of the capillaries of the kidney by the debris of lysed red cells and of the tubules by cellular debris and casts. It is now known to be attributable to tubular damage from severe anoxaemia. The effects are accentuated by the acute haemolysis which produces vascular changes in the renal cortex and further damages the tubules.

Symptoms

188. *In severe cases* due to the inhalation of a relatively high concentration of the gas, symptoms develop rapidly—shivering, headache, pain in the epigastrium and over the kidneys, vomiting, breathlessness, weakness and giddiness, the patient rapidly becoming comatose, with a steady weakening of the pulse. Evidence of serious destruction of the red corpuscles of the blood is soon afforded by the passage of coffee-coloured urine. In the worst cases, oliguria occurs immediately after exposure and anuria follows very rapidly. Cases with severe oliguria and anuria are usually fatal. If the patient survives, intense jaundice accompanied by enlargement of the liver soon ensues.

189. *In cases of moderate severity* the symptoms are of the same general type—shivering, weakness, giddiness, nausea and vomiting, headache often associated with insomnia, renal and hepatic pain, dyspnoea on exertion and diarrhoea. Headache and repeated vomiting seem to be almost constant features. Haemoglobin soon appears in the urine, which also shows albumin and casts. Jaundice develops a little later.

190. *In mild cases* anaemia may develop gradually with a progressive fall in the red cell count and the haemoglobin content of the blood, without the appearance of haemoglobin in the urine and with little or no jaundice. This anaemia is associated with the symptoms of lassitude, headache, general malaise and undue breathlessness on exertion.

At a later stage arsenical neuritis, particularly of the extremities, occasionally develops, manifested by pain along the line of the nerve trunks, and by some diminution of sensory appreciation.

Diagnosis

191. Haemolysis may be recognized by making equal dilutions of the patient's blood and of normal blood, and comparing the supernatant fluids after sedimentation. The following technique is convenient:—

Prick the finger of the patient and allow 3 drops of his blood to fall into 12 drops of a 3·8 per cent sodium citrate solution in a watch glass or small tube.

Mix well and draw up the mixture into a glass tube of length 20 cm. and internal bore 2-3 mm., the end of which has been slightly tapered as for a pipette. Close the lower end of the tube with a short length of rubber tubing which has been stoppered with a piece of glass rod or clipped.

Prepare a precisely similar tube with the blood of a normal person to serve as a control.

Stand both tubes vertically for half-hour or longer so that sedimentation of the corpuscles may take place.

If there is any haemolysis in the suspected blood it will be shown by the tint of the supernatant fluid as compared with that of the control.

Differential diagnosis.—Blackwater fever, paroxysmal haemoglobinuria and other forms of acute haemolytic anaemia, and also acute nephritis from other causes may be confused with arsine poisoning.

Protection

192. Untreated charcoal is an inefficient absorbent for arsine. A small quantity of silver is therefore used to impregnate the charcoal of modern respirator containers, and by this means complete protection is obtained.

Treatment

193. *(a)* In severe cases, such as those likely to prove fatal within 48 hours of exposure, oxygen under pressure is probably the only measure likely to be of value. It can be given through an oro-nasal mask using a flow of 5-6 litres of oxygen per minute.

(b) Intramuscular injection of BAL has been used to counteract the systemic effects of arsenic poisoning and to assist in the elimination of arsenic from the body. In arsine poisoning BAL may give some protection if given before exposure or within six hours of exposure.

(c) *Blood transfusion* is indicated whenever the haemoglobin falls below 50 per cent, or red cells below 2·5 million per cubic mm. The risk of haemolysis of the transfused blood by arsine is slight after an interval of 2 to 3 hours after exposure, but transfusion may need to be repeated in a few cases since haemolysis may continue for several days.

(d) *Administration of glucose.* Glucose should be given freely from the onset, either with or without insulin. It is especially indicated if there is evidence of necrosis of the liver when, if necessary, it may be injected intravenously. It is also very useful in anuria as it suppresses protein destruction and keeps the blood potassium level low.

(e) *Renal failure.* In every case of appreciable severity there will be evidence of defective renal function as shown by the reduction in the quantity of urine excreted, possibly ending in anuria. Blood urea will rise progressively. There is also a change in the electrolytic balance, with hypochloraemia, a lowering of the alkali reserve, and progressive potassium retention. The serious disturbance of electrolyte fluid balance results in water retention. No simple therapy is capable of counteracting these vital changes; even in the most favourable circumstances treatment is a difficult problem. The subject is too vast to be dealt with in detail in this manual, but the treatment of anuria as suggested by Bull, Joekes and Lowe is the most practical that can be recommended until the kidneys recover sufficiently to resume their normal function.

Their suggested treatment consists of a daily diet of glucose 400 gm. and peanut oil 100 gm., emulsified with tragacanth and made up to 1 litre with distilled water. This is administered by intragastric drip using a plastic tube passed intranasally. Any vomitus is filtered and returned through the stomach tube in order to conserve electrolytes. In a temperate climate the only additional fluid given is water equal in volume to the amount of urine passed daily. Where there is much sweating extra fluid must be given to compensate for the loss through the skin. A very useful check on the fluid is to weigh the patient twice daily. Any loss of weight in 24 hours is made up by additional fluid intake, whereas any progressive increase in weight suggests overhydration and a cut in fluid intake. During the recovery phase, when the kidneys begin again to excrete urine, water balance must be maintained by increasing the fluid by mouth. The gastric drip must then be stopped and a fruit diet instituted, or else the patient will develop gross potassium insufficiency. At this stage too it is just as important to maintain electrolytic balance, as a deficiency due to profound diuresis is liable to occur.

(f) *Special risks.* In the wounded, watch must be kept for secondary haemorrhage. In choosing anaesthetics, the presence of liver damage needs consideration.

(g) *Convalescence.* Light diet is indicated in view of the liver and kidney damage. Anaemia must be treated and cases should be kept under observation until both blood and urine are normal.

(h) Antibiotics should be given to all patients with anuria. Penicillin 1,000,000 units initially intramuscularly, and 250,000 daily will maintain a high blood level. Alternatively, Aureomycin or Terramycin 1·5-2·0 gms. the first day, and then 250 mgms. *twice weekly* for the duration of the anuria.

194-200. *Reserved.*

CHAPTER X

CARBON MONOXIDE

201. Carbon monoxide is a colourless, odourless non-irritant gas which cannot be recognized by the senses. It is formed wherever combustion of carbonaceous material is incomplete, and is therefore commonly met under normal conditions of everyday life. It burns with the characteristic blue flame so often seen flickering over a coke or smouldering coal fire.

Occurrence

202. Carbon monoxide is always present in dangerous amounts in the exhaust gases of internal combustion engines, coke stoves or smouldering fires, while varying quantities of it are present in all types of illuminating gas; it also forms the deadly constituent in the so-called " after-damp " in collieries.

In war, carbon monoxide may be met in dangerous quantities under the following conditions:—

(a) Mining operations.—Whenever a blasting charge is exploded, as in mines or camouflets, the resulting gases contain large quantities of carbon monoxide, which are liable to find their way to adjacent galleries, trenches or dug-outs; moreover, pockets of gas may occur which may be tapped when new galleries are being driven in the vicinity. Carbon monoxide is also produced in large quantities when the timbering of mine galleries catches fire.

(b) Heavy gun fire.—A high-explosive shell penetrating the soil, and bursting in close proximity to confined living quarters such as dug-outs, may liberate sufficient carbon monoxide to poison the occupants.

(c) Gun emplacements, pill-boxes and ships' turrets.—Carbon monoxide gas may accumulate rapidly in gun emplacements, pill-boxes or turrets of ships, especially when firing into the wind, owing to the blow-back of muzzle gases or when the breech of the gun is opened; in the case of ships' turrets, if the air-blast in the barrel be defective, high concentrations of the gas may accumulate within the turret.

(d) Underwater explosions.—At sea, following underwater explosions, large quantities of carbon monoxide may be trapped within the hull of the ship and may gain access to inner, inhabited compartments.

(e) Interior of tanks.—If ventilation be defective, dangerous concentrations of carbon monoxide may be found in tanks from leakage of engine exhaust gases and from blow-backs from the guns.

(f) Burning buildings.—Carbon monoxide gas is generated by the burning of material in closed spaces, owing to insufficiency of oxygen.

(g) Coke and charcoal fires.—Poisoning has often occurred through the use of coke or charcoal braziers or stoves in insufficiently ventilated billets.

(h) Internal combustion engines.—The exhaust gases of internal combustion engines contain from 1 to 10 per cent or more of carbon monoxide; the use of such engines in confined spaces without adequate safeguards may have serious results.

(i) Aircraft.—Dangerous concentrations of carbon monoxide may occur in some types of piston engined aircraft. This, however, is unlikely to occur in pressure cabined or jet propelled aircraft.

(j) Fractured gas mains.—The leakage of coal gas into closed spaces, such as billets, following fracture of a gas main (*e.g.*, after a bombardment) may cause poisoning.

Mode of Action

203. Carbon monoxide owes its poisonous properties to the fact that it combines with haemoglobin to form a dissociable compound, carboxy-haemoglobin. Its affinity for haemoglobin is more than 200 times that of oxygen. But for this property of combining with haemoglobin, carbon monoxide would be a physiologically inert gas like nitrogen or hydrogen.

When air containing carbon monoxide is inhaled, the relative amounts of oxygen and carbon monoxide present in the atmosphere determine the proportion in which carboxy-haemoglobin is found in the blood. When the amount of oxygen is 200 times that of carbon monoxide, half of the haemoglobin can combine with the carbon monoxide and half with the oxygen; this is about the degree of blood saturation at which unconsciousness occurs.

As the concentration of the gas in the air rises, the saturation of the haemoglobin with carbon monoxide increases, and the oxygen-carrying capacity of the blood progressively diminishes until symptoms of anaemic anoxia (oxygen want, in this instance without cyanosis) make their appearance. In this sense the gas is cumulative in action (*see* anaemic anoxia, para. 251).

Moreover, the rate of absorption of carbon monoxide is very much accelerated by muscular exertion or by mental excitement, which causes an increase in the breathing and circulation rates. This results in a more rapid diminution in the available oxygen content of the blood, with a corresponding increase in the symptoms of oxygen want.

The consequences are due to anaemic anoxia and in severe cases to a direct toxic action on the nerve cells. There are no pathological changes in the lungs such as follow the action of asphyxiant gases, nor are the red blood corpuscles injured; when freed from their combination with carbon monoxide, the corpuscles are as capable of resuming their normal function as oxygen carriers as they were before exposure to the gas.

Death occurs when saturation of the haemoglobin reaches about 70 to 75 per cent, but lower degrees of saturation of the blood may prove fatal if exposure to the poisonous atmosphere is prolonged. The colour of the blood and tissues, post mortem, may be bright red.

Symptoms

204. The great danger in carbon monoxide poisoning is the risk that it may not be suspected until too late. Early symptoms may be a loss of power in the limbs, which makes escape difficult or impossible.

Where the proportion of carbon monoxide to oxygen is high, loss of consciousness may be very rapid, with practically no warning. More commonly, however, the onset is gradual and insidious. The first symptoms may be a

feeling of weakness, giddiness, vomiting and indistinct vision; these are followed by breathlessness, palpitation and a loss of power in the limbs. The least exertion at this stage may cause collapse.

Experienced pilots are aware of the danger due to leakage of carbon monoxide in aircraft, although they may not recognize the early symptoms, such as loss of brightness perception, and the fine co-ordination of the ocular muscles.

The loss of muscular power and the confused cerebration often prevent a man from withdrawing from danger even though he is dimly aware that safety is only a few yards distant. Not infrequently there is a stage of acute mental excitement, which may simulate alcoholic intoxication or even mania. This is more common in the milder cases. Apathy and a sense of complete helplessness supervene, followed by unconsciousness, with or without convulsions; the victim becomes comatose, with stertorous breathing, a low-tension pulse and subnormal temperature, and death results if he be left in the poisoned atmosphere.

The colour of the face may vary with the rapidity of the onset or the degree of anoxaemia. A leaden tint is often seen after profound coma, while in other cases the face may be pale and moist with perspiration; often, however, the cheeks are pink and the lips a vivid carmine.

Individual susceptibility to the gas varies, and experience has shown that acute or chronic alcoholism, and cardiac or respiratory disorders, accentuate the severity of carbon monoxide poisoning.

Recovery from the initial symptoms may be followed by mental confusion and slow cerebration, while headaches, often of a severe or migrainous type, are characteristic.

Severe carbon monoxide poisoning *per se* can produce a state of hypotension. This is due to cardiac failure from lack of oxygen and may be preceded by a slowed pulse due to heart block. Venous blood in carbon monoxide poisoning is not reduced and is bright red in colour. Often there is no pallor, especially in the early stages, but there is sweating despite a fall in body temperature. The history of exposure to explosive or exhaust gases in a confined space—and the accompanying features such as mental confusion, glycosuria, patchy pulmonary atelectasis—help to form a diagnosis. Among other after-effects may be mentioned cardiovascular disorders, especially tachycardia and dyspnoea which may continue for months, and a predisposition to pneumonia. Disturbances of the central nervous system range from a simple neuritis to paresis and even mental derangement, usually of temporary duration, but sometimes the result of damage to the brain cells by protracted anoxaemia. Mental relapse after apparent recovery is of serious significance.

Protection

205. *The respirator does not afford any protection against carbon monoxide.* If it is necessary to enter an atmosphere in which the gas is present or suspected, it is essential that a special carbon monoxide respirator* be used (*e.g.* mask with filter consisting of catalysts which oxidize the carbon monoxide to the dioxide) or some form of self-contained oxygen-breathing apparatus such as the Proto or Salvus set, or, in the case of H.M. ships, the Davis S.E. apparatus, which is more familiar to the personnel.

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* Carbon monoxide respirators must be used with great care since oxygen deficiency is generally associated with the presence of carbon monoxide, and these respirators give no protection against this danger.

In the absence of special oxygen sets, a useful apparatus can be readily extemporized by means of an ordinary respirator facepiece to which is attached a suitable length of non-collapsible tubing of 1·5-inch diameter, the far end of which is left out in the open. Such an apparatus can only be worn for short periods, owing to the absence of an inlet valve and the rapid accumulation of carbon dioxide in the tubing; the insertion of a valve, however, at the inlet end of the facepiece valve holder will enable a man to remain in a contaminated atmosphere almost indefinitely. Such an apparatus (with special valve) is standardized in H.M. Navy, and is known as B.A. Pattern 230. A similar remote breathing apparatus is available in the R.A.F. under the stores reference number 21F/611.

Small animals, such as mice or canaries, can serve as indicators of the presence of carbon monoxide Owing to their rapid metabolism, they show signs of poisoning before man is affected.

Test for the Detection of Carbon Monoxide in Blood

206. The simplest method of detecting carbon monoxide in the blood is as follows: Dilute a sample of the suspected blood in a test tube (of internal diameter of about 1·5 cm.) to 0·5 per cent with distilled water to which a trace of ammonia has been added (*e.g.* 0·1 c.c. blood diluted to 20 c.c.); this will give a perfectly clear solution. Dilute similarly a sample of normal blood.

Compare the tints of the two test tubes by transmitted daylight, when it will be seen that the tint of the suspected blood, if carbon monoxide be present, is definitely more pink than the yellowish-red of the dilute normal blood.

This test is purely qualitative; the actual saturation in any suspected specimen can be determined either by the carmine method or by means of the reversion spectroscope.

Treatment

207. The majority of cases of carbon monoxide poisoning recover with prompt treatment, although relapse, or even sudden death, is possible later. Unconsciousness may last as long as 48 hours after regaining pure air and yet the person may recover; but the longer unconsciousness lasts the less is the chance of recovery.

Treatment consists in the prompt administration of oxygen, aided by artificial respiration which may have to be continued for some hours. Some authorities advocate the use of oxygen/carbon dioxide mixtures but the advantages of this over pure oxygen remain controversial. The patient should be kept warm.

Oxygen displaces carbon monoxide far more rapidly than ordinary air. It should be stressed that the expired air must not be re-breathed, and with this in view a suitable apparatus should be used such as the B.L.B. (*see* Chapter XIV), which may be set to deliver 8 to 10 litres per minute.

A characteristic effect of carbon monoxide poisoning is a lowering of the body temperature, due to a disturbance of the heat regulating centre and to a reduction in the normal oxidative processes. Even in mild cases patients may complain bitterly of cold, and it is necessary that this symptom be combated by means of hot coffee, blankets, hot-water bottles and other familiar measures. The patients often sweat and inexperienced attendants may remove extra clothing: nevertheless they are cold, remaining so for many hours, and need considerable warmth.

Rest, too, is imperative in order to avoid any increase in the oxygen requirements of the body and to reduce the demands on the ill-nourished heart.

During convalescence, especially after severe or prolonged anoxaemia, particular care should be taken that no great strain be thrown on the heart, owing to the risk of acute dilatation.

208-212. *Reserved.*

CHAPTER XI

MISCELLANEOUS GASES

(A) Chlorine

213. Chlorine gas was the first chemical agent to be used in modern war. It was released from cylinders by the Germans on the Western Front in World War I.

It is a yellowish-green gas and is easily seen when liberated from cylinders, bombs, etc. Chlorine has the pronounced smell of bleaching powder and is highly irritant to the mucous membranes. Because of its colour and its irritant effects it is quickly recognized and by putting on the respirator early, complete protection is obtained.

Chlorine gas has no action on the skin but when inhaled is highly irritant to the muccus membranes of the upper respiratory passages, causing a violent and paroxysmal cough which may persist for some time after exposure. It is less toxic than phosgene but may cause serious damage to the trachea and bronchi, the epithelial lining being severely affected and desquamation taking place. The tendency to blockage of the respiratory passages is therefore great and the paroxysms of violent coughing produced by chlorine tend to induce a disruptive emphysema which is much more marked in chlorine than in phosgene poisoning.

Subcutaneous emphysema was rarely seen in World War I except after gassing with chlorine.

(B) Fumes Encountered in Fire Fighting

Noxious products of combustion

214. Fire fighting, especially when practised inside buildings or in confined spaces, often entails risk, either because of noxious gases or through a deficiency of oxygen; hence the various protective devices used by firemen, such as respirators and oxygen-breathing apparatus.

In all fires in confined spaces the nature and concentration of the toxic gases produced vary with the rate of combustion and with the character of the burning material. Thus, a slow rate of combustion results in a heavy concentration of carbon monoxide and carbon dioxide, in addition to an oxygen deficiency, while burning cordite (as in a magazine) gives rise, in addition, to nitrous fumes.

When chemical extinguishers are used to quell fires in confined spaces, additional toxic gases may be produced, causing further danger to unprotected men.

The unfailing presence of carbon dioxide hastens the onset and increases the severity of any toxic symptoms that may result. Carbon dioxide is more than a simple asphyxiant in that, at comparatively low concentrations, it causes increased breathing and thereby increases the quantity or dose inhaled of any noxious gas that may be present. In concentrations above 10 per cent it produces unconsciousness and death.

The utility of the ordinary anti-gas respirator in fire fighting is strictly limited, and is confined to the arrest of particulate products of combustion and of such gases as can be dealt with by the charcoal in the container. These do not include either carbon monoxide or carbon dioxide. Further, as the respirator cannot compensate for oxygen deficiency, it is essential that, when fighting fires where a free dilution of the atmosphere or a free escape for noxious gases is not possible, an oxygen or air breathing apparatus should be worn.

Chemical fire extinguishers

215. The following are two types in common use, sold under various trade names:—

(a) Carbon tetrachloride.—This is a volatile liquid, boiling at 76·7° C. (170° F.) which is extensively employed as a dry-cleaning agent and as a popular and effective fire extinguisher.

When carbon tetrachloride is sprayed on a fire or on a heated surface the chief decomposition products, in addition to the unchanged chemical, are phosgene, hydrochloric acid and chlorine. The production of phosgene is greater when the liquid comes in contact with heated rusty iron and when large quantities of the extinguisher are used in the presence of moisture.

Although the thermal decomposition products are more or less irritant to breathe, this irritancy may not be such as to compel men, faced with a dangerous emergency, to leave a burning room. In these circumstances a very real danger arises from the continued inhalation of vaporized carbon tetrachloride or its products.

Recent experience has shown that exposure to the fumes of carbon tetrachloride in a confined space such as a garage or between decks may give rise to serious illness, often delayed in its onset, of renal and hepatic origin. The illness may be ushered in by pyrexia, general malaise and abdominal pain—a commonplace clinical picture which may lead to errors in diagnosis.

Personal idiosyncrasy plays a part in the character, as well as in the severity, of the resulting symptoms; but, as a rule, signs of impaired kidney function are always present, varying from a trivial rise in blood pressure to an acute uraemia. Evidence of liver damage may also be seen in the jaundice, the slow pulse, the abdominal pain and haemorrhage from stomach and bowel so characteristic of the toxic jaundice caused by the organic halogens.

(b) Methyl bromide.—Another type of fire extinguisher contains methyl bromide as its chief constituent. This is a gas at ordinary temperatures, but it is readily liquefied at 0° C., to a clear, colourless and extremely volatile liquid which boils at 4·5° C. (40·1° F.). The gas is almost odourless.

Methyl bromide is toxic, and its thermal decomposition products are practically irrespirable. The liability of this extinguisher to produce poisoning, however, is chiefly determined by the rapid rate of volatility of the undecomposed chemical, which is much higher than that of carbon tetrachloride. Owing to its rapid vaporization a toxic dose may be inhaled before the danger is appreciated.

In high concentration methyl bromide has a profound effect on the central nervous system, producing unconsciousness, epileptiform seizures and paralysis, both motor and sensory.

In less severe cases vertigo, visual troubles and general weakness are the usual symptoms, and it does not appear that the dose need be large to produce them.

(C) Fumes Associated with Nitro-explosives

216. When nitro-explosives are incompletely detonated or subjected to slow combustion, especially in confined spaces, considerable quantities of " nitrous fumes ", consisting of a mixture of oxides of nitrogen, are given off.

These fumes, which have an orange-yellow or reddish-brown colour, are very soluble in water, and react readily with moisture and oxygen to form nitric and nitrous acids. In damp surroundings, therefore, the concentration of these gases in the atmosphere will be lowered.

Occurrence

217. Under war conditions, nitrous gases may be met with in mining or tunnelling operations when detonation of the blasting charge is incomplete, in gun pits, armoured cars, and tanks, and in magazines of ships when propellant charges are set on fire. In industry, dangerous concentrations may be evolved when nitric acid is heated, or when it comes in contact with organic material, such as wooden floors, after accidental spilling.

Although no serious cases of poisoning by these gases were recorded on land in World War I, this may have been partly due to the fact that when nitrous fumes are formed in large quantities in mining operations, carbon monoxide, with its more rapid action, is also generated in lethal proportions. Another possible factor was the moist condition of the surroundings, which may have helped to reduce the concentration of nitrous gases.

At sea, the virulence of the nitrous gases was well illustrated in World War I by the death-rolls which followed the sinking of H.M. Ships " Russell " and " Britannia," when a large proportion of the officers and men who had been exposed to the fumes of burning cordite succumbed to their effects.

Mode of action

218. The action of nitrous fumes on the lungs closely resembles that of phosgene. They are particularly dangerous because they do not produce much sensory irritation, and men may therefore fail to realize the serious danger which may follow their inhalation.

When inhaled, the nitrous fumes come into contact with the moisture ever present in the respiratory tract, and form nitric and nitrous acids; this produces a local caustic effect, to which is superadded a general systemic action due to absorption of the alkaline nitrites formed by the interaction of the acids with the alkaline secretions in the presence of oxygen.

As with phosgene, the local action gives rise to an intense congestion of the lungs, with inflammation and oedema. This usually overshadows the systemic effect of the alkaline nitrites, which, however, contribute to the clinical picture by their enfeebling action on the circulation. Through the formation of methaemoglobin, they may also diminish the oxygen-carrying capacity of the blood.

With nitrous fumes, as with phosgene, the initial symptoms of coughing and irritation are generally transitory, and a period of quiescence precedes the onset of the acute symptoms. This may vary from 2 to 24 hours or more, according to the conditions of exposure, its usual duration being between 10 and 24 hours. Once this period is over, the clinical signs develop rapidly, and the whole course of a possibly fatal illness may be run in a few hours.

Symptoms of poisoning

219. The symptoms on exposure are slight irritation of the eyes, nose and throat, accompanied perhaps by a little cough—symptoms which are seldom at all conspicuous and which quickly subside during the latent period which follows. The termination of this latent period, which may be precipitated by physical exertion, is marked by the onset of acute clinical signs and symptoms such as a dry, hacking and painful cough, a sense of constriction in the chest, and distressing breathlessness.

In mild cases, this may be a prelude to a bronchitis which is limited to the upper bronchi and is associated with a profuse mucopurulent expectoration. In more severe cases, however, a condition of acute bronchial spasm may set in, with pulmonary congestion and cyanosis, rapidly followed by a pulmonary oedema which may be haemorrhagic in character. Restlessness is extreme, and in fatal cases consciousness is retained almost to the end, the patient struggling vainly for breath while, with bloodstained fluid trickling from his mouth and nostrils, he drowns slowly in the fluid exuded in his lungs.

Protection

220. The ordinary charcoal respirator affords limited protection against nitrous fumes, but none against the carbon monoxide which is nearly always generated simultaneously. It is essential, therefore, that an oxygen or air breathing apparatus (such as the Proto, Salvus, D.S.E.A., the Naval B.A. Pattern 230 or the R.A.F. Pattern 21F/611.) be worn in such atmospheres whenever possible, and reliance should not be placed on a respirator save in emergency.

Nitrous fumes may be readily demonstrated by means of test papers which have been previously dipped in a solution of starch and potassium iodide and slightly acidified; a blue coloration develops on them when they are exposed to the gases.

Treatment

221. Treatment follows the same lines as those outlined for phosgene poisoning, stress being laid on complete physical rest from the time of exposure and on administration of oxygen as soon as cyanosis develops. Broncho-pneumonia is a complication in some cases.

Convalescence is apt to be prolonged, and the experience of World War I showed that the combined action of carbon monoxide and the nitrous fumes had a harmful effect on the heart, necessitating careful surveillance and graduated exercises.

(D) Gasoline

222. Gasoline varies very considerably in composition: it consists of a mixture of saturated and unsaturated hydrocarbons to which are added anti-detonant substances such as benzene, alcohol, or tetraethyl lead.

Gasoline fumes are toxic to humans in concentrations over 1 per cent, unless exposure is short. Engineers, mechanics and cleaners are most liable to risk. Dangerous concentrations may be encountered in tanks, wagons or compartments which contain or have contained gasoline.

Tetraethyl lead is present in solution in certain types of spirit. It is a toxic liquid which is readily absorbed through the skin, and presents a particular danger in the " slush " which settles in tanks, etc. Gasoline containing tetraethyl lead should not be used for cleaning the hands.

Men who have to enter containers or compartments containing appreciable amounts of gasoline vapour should be protected by airline respirators or self-contained oxygen apparatus and suitable clothing. They should be in touch with a sentry outside the tank, and the sentry should be equipped for rescue work.

Current opinion in industrial and service practice is against the use of G.S. and light type respirators, which do not afford much protection:—

against $1\frac{1}{2}$ per cent concentrations: about 20 minutes,

against 7 per cent concentrations: about 5 minutes.

A pronounced smell indicates a concentration over 1 per cent. In conditions necessitating violent exercise (*e.g.*, rescue work), the time factor is reduced by about 50–90 per cent. The smell of gasoline within the facepiece will indicate that protection is being lost.

Effects

223. Gasoline vapour in high concentrations is a powerful anaesthetic which causes, besides irritation of throat, nose and eyes; headaches, dizziness, drowsi-ness and loss of consciousness. Lower concentrations produce the same effects, though more slowly. General convulsions, respiratory failure and collapse may follow the earlier symptoms. The margin between the dose necessary to produce coma and that causing death is narrow. If the victim recovers from the coma he may develop a state of delirious excitement and violence.

224. Absorption of tetraethyl lead causes cortical effects, the most important of which are restlessness, insomnia and nightmares. Talkativeness, tremors, ataxia and delusions also occur. These symptoms may be superimposed on those due to gasoline vapour.

225. If gasoline is splashed on bare skin it quickly evaporates and no skin burns result: if however clothing is saturated with gasoline, prolonged contact with the liquid results and blistering of the skin takes place. It is usual to find extensive burns over the entire back or other parts of the body which have been in contact with liquid gasoline in persons who have collapsed in an enclosed compartment.

Treatment

226. When a man has collapsed in a gasoline laden atmosphere, speedy treatment is essential. It should be remembered that gasoline vapour is heavier than air, and the victim may have fallen into a heavier concentration than that which caused his collapse. The mixture may also be explosive. The patient should be brought to the fresh air by a rescuer, suitably protected. Gasoline-soaked clothing should be removed and the skin be washed as soon as possible. Artificial respiration should be administered if necessary.

227-230. *Reserved.*

CHAPTER XII

SMOKES AND INCENDIARY SUBSTANCES

Screening smokes

231. Smoke may be used for screening important positions or the movement of troops; it may also be employed to mask a gas cloud, or to extend its flanks so as to conceal its actual frontage. Such screening smokes may be generated from solids dispersed from shell or bombs, or from liquids sprayed from aircraft or land vehicles.

Screening smokes are irritating when inhaled in close proximity to their source, but they are not toxic in the concentrations that render them effective as screens; under ordinary conditions troops can operate in them without wearing respirators, while in higher concentrations they may be irritant yet not produce toxic effects.

A dangerous and possibly asphyxiating concentration, however, might arise if a smoke shell bursts at, or close to, the entrance of a dug-out, while proximity to a bursting phosphorus smoke shell may result in very severe burns from flying particles of burning phosphorus (*see* para. 236).

Apart from these possibilities, the chief danger associated with the use of screening smokes arises through accidental contact with the chemicals used in their production. These chemicals are all corrosive or dangerous to handle, and accidental contamination of the eye or splashes on the skin with the liquids will cause severe ulceration or burns. With a view to preventing such accidents, operators should wear protective goggles or respirators, thick gloves, rubber boots, and special coats of oilskin or rubber.

The respirator gives efficient protection against all the screening smokes, and clothing is not affected by exposure to them in the concentration met with in the open.

Chlorosulphonic acid (C.S.A.)

232. This is a fuming, highly corrosive liquid which, on contact with quicklime, gives off a thick white cloud closely resembling a dense mist. At close quarters this irritates the eyes and throat sufficiently to necessitate the wearing of a respirator, but at a distance of 200 yards or more from the source of emission this can easily be dispensed with.

Owing to its highly corrosive nature, C.S.A. requires great care in handling; moreover, in contact with water C.S.A. generates intense heat, and acid may be scattered in all directions. Treatment follows the convential methods adopted for any corrosives.

Titanium tetrachloride (F.M.)

233. This is a yellow, non-inflammable and corrosive fluid which, on contact with damp air, gives off a heavy dense white cloud. This property is made use of by aircraft for the production of vertical smoke curtains extending down to the ground or sea level. The smoke consists of fine particles of free hydrochloric acid and titanium oxychloride, and its efficiency depends largely on the amount of moisture present in the air.

The smoke is unpleasant to breathe, but it is not toxic; the wearing of goggles or a respirator, however, may be necessary when entering a smoke curtain if the spray is still falling, owing to the danger of drops entering the eye. The usual precautions must be taken when handling the liquid, and contamination of the eyes or of the skin should be treated by conventional methods adopted for any corrosives.

Stannic chloride (K.J.)

234. Stannic chloride is a fuming, straw-coloured, corrosive liquid which produces a heavy white cloud on contact with air, and is therefore sometimes utilized by aircraft for the production of vertical smoke curtains. The dangers attending the handling of it are similar to those associated with titanium tetrachloride, and the treatment is the same as for other corrosives.

Zinc chloride

235. Certain mixtures in use, *e.g.*, Berger type smoke mixtures, produce zinc chloride in the form of a vapour which condenses and combines with moisture to form a particulate cloud.

In the field the concentration normally encountered in a smoke screen is insufficient to be dangerous, but it must be remembered that in high concentrations the fumes constitute a serious hazard. Such conditions may arise in the immediate neighbourhood of bulk stocks which have become accidentally ignited.

The respirator gives protection against high concentrations for only a short period.

Zinc chloride smoke causes smarting of the eyes and irritation of the respiratory tract, with coughing, bronchial spasm, and laboured respirations. Nausea and syncope occur, and the worst cases develop pneumonia and pulmonary oedema.

Adrenaline may be given in the acute stage of bronchial spasm, and oxygen may be necessary. When the exposure has been heavy enough to cause systemic absorption, BAL given by intramuscular injection is recommended.

Phosphurus

236. At ordinary temperatures white phosphorus is a solid which can be handled safely in water but, when dried in air, burns fiercely with a dense white smoke. It may be used by an enemy as an incendiary or as a smoke filling in bombs or in shell, and flying fragments or melted particles of the burning substance may become embedded in the skin of persons close to the bursting missile. These fragments continue to burn unless flooded or smothered. Burning clothes may be ripped off and flames extinguished with water, blankets, sand or earth.

237. First aid treatment should consist in immersion of the affected part in water, or, in the absence of enough water, in the application of a thick pad soaked in water. This purely temporary measure should be followed at the earliest opportunity by removal of all phosphorus from the skin, according to the following directions:—

(a) Bathe the affected part in sodium carbonate or bicarbonate solution (two tablespoonfuls of washing soda or bicarbonate of soda to the pint of water) for a few minutes, to neutralize phosphoric acid, which is formed when phosphorus burns on the skin, and to allow of the removal from the skin of any visible phosphorus. Fragments otherwise unobserved may be seen in the dark.

(b) The area should be washed with copper sulphate soap if available; otherwise immerse the injured part in a solution of copper sulphate (one heaped teaspoonful to a pint of water) for some seconds. All dark-coloured deposits which are formed by the action of the sulphate or any remaining phosphorus should then be removed, with the aid of forceps, in clean water. If this removal is incomplete and free phos-

phorus is left, even in minute amounts, on the skin—detected by fuming and the typical smell of white phosphorus—these two stages should be repeated.

After these preliminary measures, further treatment should be as for any other thermal burn.

Ultra-violet light is of value in the treatment of phosphorus burns. More rapid healing is promoted, and the great pain always associated with skin lesions due to phosphorus is lessened. Daily exposures of 1 to 1½ minutes' duration should be given from the very beginning of the treatment.

238. If the eyes are affected treatment should be by lavage, except that where practicable initial irrigation should be performed with a 1 per cent solution of copper sulphate. Water, saline or any bland fluid is suitable, but it must be used without delay. If it is impossible to open the lids, no time must be lost in securing skilled assistance, so that the lids may be parted and irrigation begun. The particles rendered visible by copper sulphate may be picked out after instillation of cocaine. Lavage should not continue after it is thought that all free chemical has been washed out. If the lesion seems to be severe, atropine should be instilled.

239. Symptoms of acute poisoning, such as restlessness, thirst, abdominal swelling and nasal discharge, may be shown by animals grazing on land contaminated by phosphorus bombs or shell. Post-mortem examination reveals fatty degeneration of the liver, and acute congestion, or even necrotic patches, in the kidneys and small intestine.

Thermite

240. Thermite (aluminium and ferric oxide) is also used in incendiary bombs and in attacks on armoured fighting vehicles.

In the first aid treatment of burns of the skin, the field or shell dressing may be thoroughly soaked with water and applied as a pad, keeping it moist. Armoured fighting vehicles are supplied with morphine (Tubunic ampoules), which should be given in all severe cases. As soon as possible any obvious particles of thermite should be removed under water. The burn should then be treated as an ordinary thermal burn.

Fuel oil and creosote

241. Burns known to be caused by fuel oil or creosote mixtures, which are sometimes used for incendiary purposes, should also be treated as ordinary thermal burns. Contamination of the skin with unignited fuel oil, however, deserves special notice, since people with highly sensitive skins (*e.g.* many of those with fair hair) may be rapidly affected. For removing the oil, soft soap is preferable to powerful solvents such as benzol and carbon tetrachloride, neither of which must be applied more than once to the same area. Gasoline is unsuitable. After cleaning, a bland unguent such as lanoline should be employed without a dressing. Hands soiled with fuel oil should never be allowed to come into contact with mucous membranes (conjunctiva, mouth, or nose).

242-250. *Reserved.*

SECTION IV—ANOXIA, OXYGEN THERAPY

CHAPTER XIII

ANOXIA AND CYANOSIS

Anoxia

251. Anoxia is a condition of oxygen lack in the tissues. This may result from:—

    (a) defective oxygenation of the blood—*anoxic anoxia or anoxaemia;*

    (b) lowered capacity of the blood for oxygen carriage—*anaemic anoxia;*

    (c) impairment of capillary circulation—*stagnant anoxia;*

    (d) impairment of tissue utilization of oxygen—*histotoxic anoxia.*

(a) ANOXIC ANOXIA OR ANOXAEMIA. In this type there is a lowered partial pressure of oxygen in the blood which in consequence is less saturated with oxygen than normally. It is produced by any circumstance which interferes with the proper oxygenation of the blood in the lungs, *e.g.*, breathing air at low pressures, as at high altitudes; by mechanical obstruction to the airway in mouth, pharynx, larynx or bronchi or by bronchospasm; mechanical interference with pulmonary interchanges, as in poisoning with pulmonary irritant gases such as chlorine or phosgene which cause pulmonary oedema; breathing air deficient in oxygen, as in closed spaces after flame thrower attacks, in the air of old tunnels and wells, closed ships' " blisters " or compartments, etc.; or by depression of the respiratory centre as in nerve gas poisoning.

(i) *Symptoms of anoxaemia* include headache, visual disturbance, mental dullness, impairment of judgment and memory, loss of muscular power and co-ordination and dyspnoea, especially on exertion. Loss of consciousness soon supervenes. Cyanosis and dyspnoea always occur in severe cases. After prolonged anoxaemia there may, as a sequel, be long-lasting damage to the central nervous system, evidenced by paralyses, loss of special senses, etc.

(ii) *Treatment.* Remove the patient to pure air at atmospheric pressure and clear airway if necessary; if respiration has stopped, artificial respiration should be applied and oxygen given if available. When the cause is pulmonary oedema, the posture should be so adjusted as to enable oedema fluid to be coughed up at intervals, and oxygen should be administered (*see* treatment of phosgene poisoning Chapter V). In all cases of anoxia care should be taken to maintain normal body temperature.

(b) ANAEMIC ANOXIA. Here the condition is the result of a diminution of the oxygen-carrying capacity of the blood from anaemia of any form or because of alteration of the corpuscular haemoglobin by carbon monoxide, nitrites and chlorates.

Carbon monoxide poisoning may be encountered in war. Here haemoglobin is converted into carboxyhaemoglobin (*see* Chapter X).

Treatment. Keep the patient warm; apply artificial respiration if necessary, preferably with oxygen. Relapses may occur even after removal to fresh air, so the patient should be watched for some hours. Convalescence may be prolonged, and if the period of unconsciousness has been long, there may be permanent damage to the central nervous system with paralyses, various types of sensory loss or loss of memory.

Arsine poisoning. The only other circumstance in which anaemic anoxia may be encountered in war would be from exposure to arsine. Arsine causes haemolysis and the conversion of the liberated haemoglobin into haematin

and possibly methaemoglobin. The symptoms, so far as is known, would resemble those of anoxaemia. Cyanosis is usually absent.

(c) STAGNANT ANOXIA. This form results from circulatory failure. The arterial blood is normally saturated with oxygen, but the circulation rate is too slow to deliver enough blood to the tissues. Hence the venous blood is much more deoxygenated than normally, and also contains an excess of CO_2. In nerve gas poisoning there may be some stagnant anoxia due to reduction in the blood flow through the lungs, although the main picture is that of asphyxia, *i.e.* a combination of anoxic anoxia and hypercarbia (CO_2 accumulation).

(d) HISTOTOXIC ANOXIA. In this condition, the flow and oxygenation of arterial blood are normal, but the tissues are unable to utilize the oxygen of the blood. The venous blood is therefore less deoxygenated than normally. The classical condition of histotoxic anoxia is seen in hydrocyanic acid poisoning, in which the oxidizing enzymes are poisoned so that the cells of all the tissues become to a greater or lesser extent incapable of utilizing oxygen.

(i) *Symptoms of histotoxic anoxia.* The symptoms are those of acute oxygen lack, with urgent dyspnoea as an early manifestation. Later, the respiratory centre is paralysed and respiration either fails or is replaced by powerful gasping breathing at a slow rate. Since the blood is not deoxygenated, there is no cyanosis, and at death the arterial blood is still fully oxygenated. Loss of consciousness occurs at an early stage, usually with convulsions, followed by paralysis and loss of reflexes.

(ii) *Treatment.* (*See* hydrocyanic acid, Chapter VIII).

Cyanosis

252. Cyanosis is due to the presence in the capillary blood of reduced haemoglobin (or sometimes other blood pigments) in replacement of oxyhaemoglobin. Cyanosis will occur when, for any reason, there is more than 5 grammes of reduced haemoglobin per 100 ml. blood in the capillaries. Normal blood contains about 15 grammes haemoglobin per 100 ml., so that if the blood is of normal corpuscular content and composition, cyanosis would be just apparent at an oxygen saturation of 66 per cent, *i.e.*, with 33 per cent or 5 grammes per 100 ml. of haemoglobin in the reduced form. Cyanosis appears, for instance, when the capillary circulation is slowed down to such an extent that the capillary blood is reduced to the requisite level of desaturation.

253. The determining condition for cyanosis is thus not the degree of desaturation so much as the absolute amount of reduced haemoglobin in the blood. Cyanosis will therefore be apparent at a smaller percentage desaturation of the blood if for any reason the total haemoglobin content is raised. This circumstance is met with when there is haemoconcentration, as for example in phosgene poisoning. At the same time, owing to the increased viscosity of the blood, the rate of flow through the capillaries is slowed, and an increased percentage of desaturation of the blood results. For these two reasons, viz. the increased haemoglobin content of the blood and the increased percentage desaturation of the blood, there is therefore a considerable increase in the absolute amount of reduced haemoglobin per unit volume of blood, so that cyanosis occurs even when the oxygen supply to the tissues is adequate. Polycythaemias in general show cyanosis for a similar reason, since, as in phosgene poisoning, the circulation rate may be so slowed by reason of the increased viscosity of the blood that even if oxygen is administered and the oxygen supply to the tissues is adequate, the cyanosis may not completely disappear.

254. In anaemic conditions the reverse state of affairs obtains, so that cyanosis may not show itself until the degree of oxygen desaturation of the blood has reached such a level as to entail severe oxygen lack. In World War I, it was common practice to perform venesection in phosgene poisoning where cyanosis was very pronounced; the resulting improvement in colour was taken as signifying circulatory improvement, but this conclusion was probably not fully justified in view of the facts mentioned above, which at that time were not known.

255. Methaemoglobinaemia constitutes another cyanotic condition, but the colour of the skin is less blue and more leaden than in deoxygenation cyanosis, and is not relieved by oxygen. Methaemoglobinaemia has occurred in toxaemia due to the wearing of certain types of impregnated clothing (anti-mustard) in hot climates, the toxic chemical being absorbed through the skin.

256-260. *Reserved.*

CHAPTER XIV

ADMINISTRATION OF OXYGEN AND ARTIFICIAL RESPIRATION

261. Administration of oxygen may be necessary for long periods in the treatment of certain pulmonary conditions accompanied by cyanosis and also in carbon monoxide poisoning. The aim should be to relieve the cyanosis, and there is no advantage in increasing the rate of flow of oxygen beyond that necessary to do this. Whilst the patient must be given enough, economy in the expenditure of oxygen must be exercised, particularly since in the forward areas and elsewhere supply may be difficult. It is desirable, therefore, that the method of administration should be efficient. Wasteful methods, such as the open glass funnel, must be avoided.

262. The following features are common to most types of apparatus for oxygen administration:—

Oxygen is supplied from cylinders of compressed gas. The cylinders are fitted with:— 1. A master tap. 2. A pressure gauge to indicate how much gas is present in the cylinder. 3. A reducing valve, to reduce the pressure to a manageable value which remains constant until the cylinder is almost empty. In some apparatus a by-pass to this valve is an additional fitting. 4. A fine-adjustment tap which may be graduated to show the rate of delivery of oxygen in litres a minute. As an alternative a bobbin flow meter may be used to measure the flow.

Several types of apparatus are in use, of which the B.L.B. Apparatus and the R.A.F. Oxygen Kit are described here.

B.L.B. Apparatus

263. This apparatus, described by Boothby, Lovelace, and Bulbulian, consists of a mask and a connecting device which joins the mask to a reservoir breathing bag (Fig. 1).

(a) *The mask.*—Two types of interchangeable rubber masks have been designed: (i) a nasal type which leaves the mouth free, and (ii) an oro-nasal type which meets the requirements of cases in which adequate breathing through the nose is impossible. All normal adults can be fitted with one or other of the two sizes, small and large, of each of these two types. The nasal mask is provided with two hollow tubes which pass on each side of the mouth and unite over the lower part of the chin into a single tube. These tubes are in open communication with the rubber bag at all times through the connecting device. The mask is held in position on the face with an adjustable rubber strap, which is placed above or below the ears according to the comfort of the patient. Slight adjustments to the shape of the mask may be made by bending the malleable strips. A small hole is provided at the base of the nasal mask for the insertion, when required, of a gastric or a duodenal tube.

(b) *The connecting device.*—On the side of the metal connecting device (Fig. 2) is an expiratory valve with a spring under slight tension, which permits the escape of any excess of expired air above that just sufficient to distend the bag, without causing any appreciable resistance to expiration. On the other side of the connecting device is an inlet connected with the oxygen cylinder; the oxygen enters through this inlet and is delivered through a tube into the lower part of the rubber bag. The upper part of the connecting device is provided with an air-regulating mechanism which consists of three small holes or ports, over which is mounted a rotating sleeve with three similar ports, one or more of which may be brought into operation by turning the sleeve. If desired, all the ports may be closed.

(c) *Reservoir breathing bag.*—The rubber bag, which is similar to a football bladder, has a capacity of about 700 c.cm. When the apparatus is in use with all the ports closed, the first part of the expired air passes from the mask through the connecting device into the rubber bag, where it is mixed with the incoming oxygen. When the bag becomes distended, the slight pressure then produced permits the excess of expired air to escape through the expiratory valve. On inspiration the expiratory valve closes and the oxygen and expired air pass from the bag through the connecting device to the mask. By adjustment of the air-regulating mechanism different amounts of atmospheric air are mixed with oxygen in the inhalation apparatus. If concentrations of 80-90 per cent of oxygen are required in the alveolar air, the apparatus must be used with all the ports closed. The flow of oxygen should be adjusted so that the bag does not collapse completely before the end of inspiration, for under these conditions the patient obtains all his inspired air from the bag. If lower concentrations of oxygen are required, one or more ports are uncovered and the patient breathes from the bag and the atmosphere.

Table to show effect of alterations in flow of oxygen and of opening or shutting ports upon concentration of oxygen in alveolar air.

Flow of Oxygen (litres per min.)	Ports open	Oxygen in alveolar air (%)	Carbon dioxide in bag (%)	Flow of Oxygen (litres per min.)	Ports open	Oxygen in alveolar air (%)	Carbon dioxide in bag (%)
3	3	46	2·04	6	0	87	1·42
4	3	56	1·51	7	0	90	0·99
5	2	69	1·22	8	0	91	0·39
6	2	76	0·89				
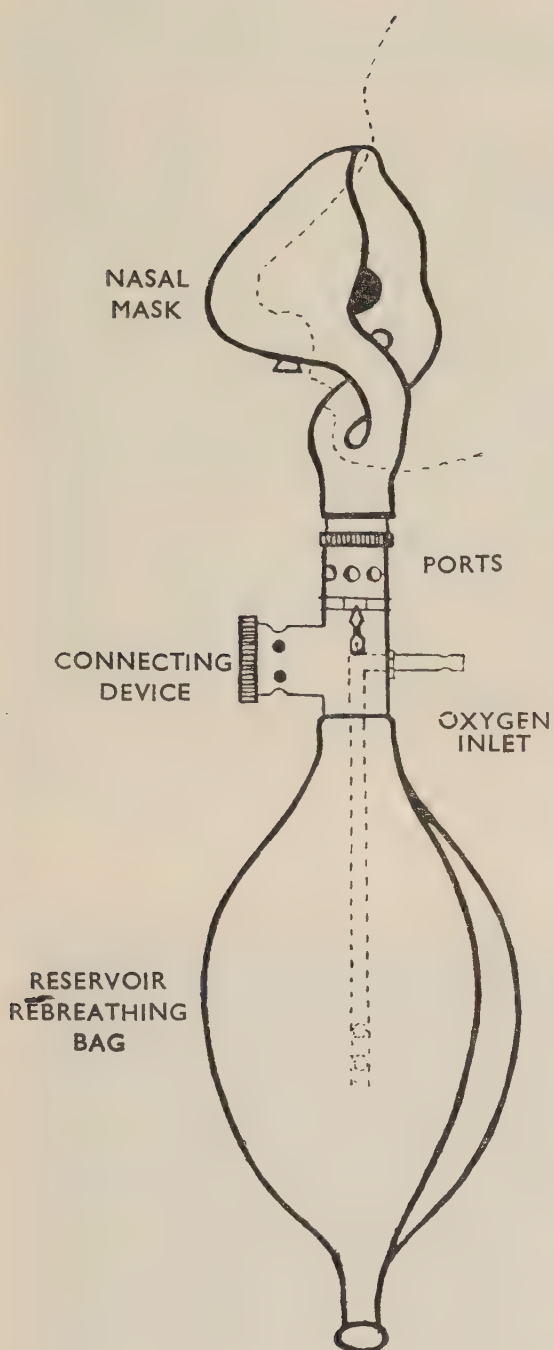
FIG. 1.—The B.L.B. inhalation apparatus.
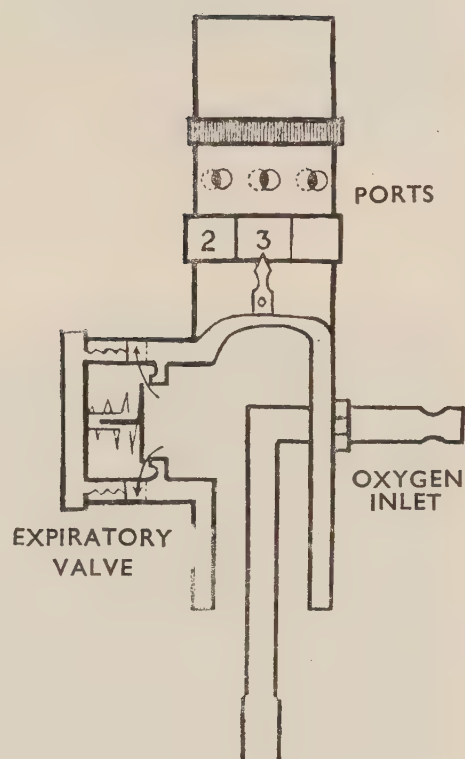
FIG. 2.—The connecting device.

R.A.F. Oxygen Administration Kit

264. This is designed as a means of emergency oxygen supply for use in ambulances, in sea rescue and in the air. One or two patients can be supplied with oxygen at the same time. If desired it can be recharged with the cylinder *in situ* and if necessary in aircraft it could even be refilled whilst actually in use.

A standard 750 litre cylinder as carried in aircraft is used, fitted with a type 7 valve. Delivery to the patient is by means of rubber tubing and a special type of expandable plastic face-piece and reservoir bag (Figs. 3, 4 and 5).

The optimum rate of flow of oxygen for one patient is 7 to 8 litres per minute, which gives a working life for one filled cylinder of 100 minutes for one patient or 50 minutes for two patients.

265. Instructions for use are fitted inside the lid (Fig. 3). They are as follows:—

(a) Recharging Oxygen Apparatus

The cylinder can be changed by undoing the yoke clamp and retaining strap. If plant is available, the cylinder may be recharged *in situ* via nipple connection on the side. This is fitted with a non-return valve.

(b) Operating Instructions for Portable Oxygen Apparatus

The mask should be applied to the face so as to be as good a fit as possible. It can be altered in shape by moulding with the fingers. The mask connecting tube must be connected to the outlet with the red ring around it when only one supply is required.

Oxygen flow should be adjusted so that the breathing bag does not quite collapse during inspiration. Flows greater than this are wasteful of oxygen and rarely (if ever) necessary and should be avoided unless specially authorized by a medical officer.
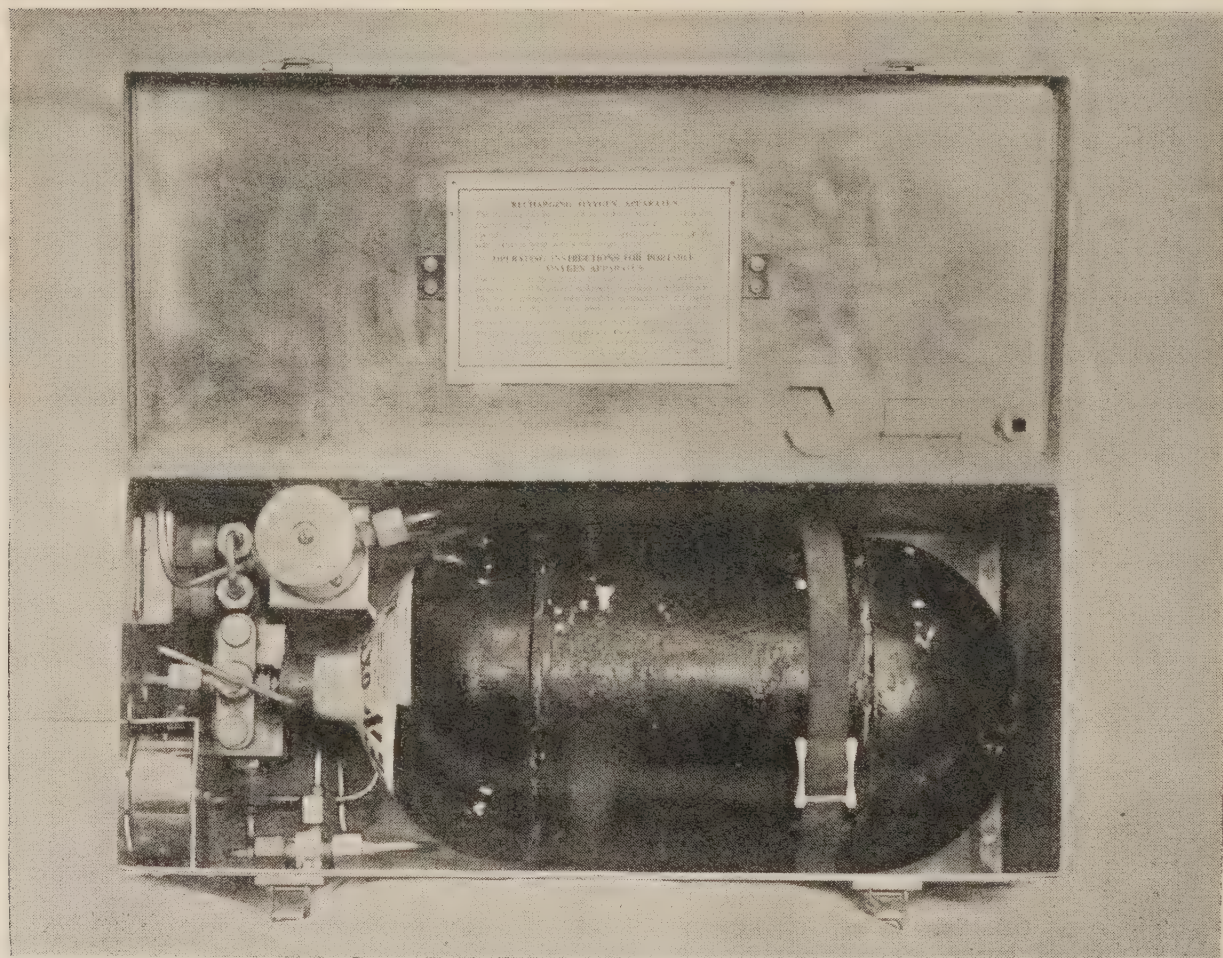
Fig. 3.
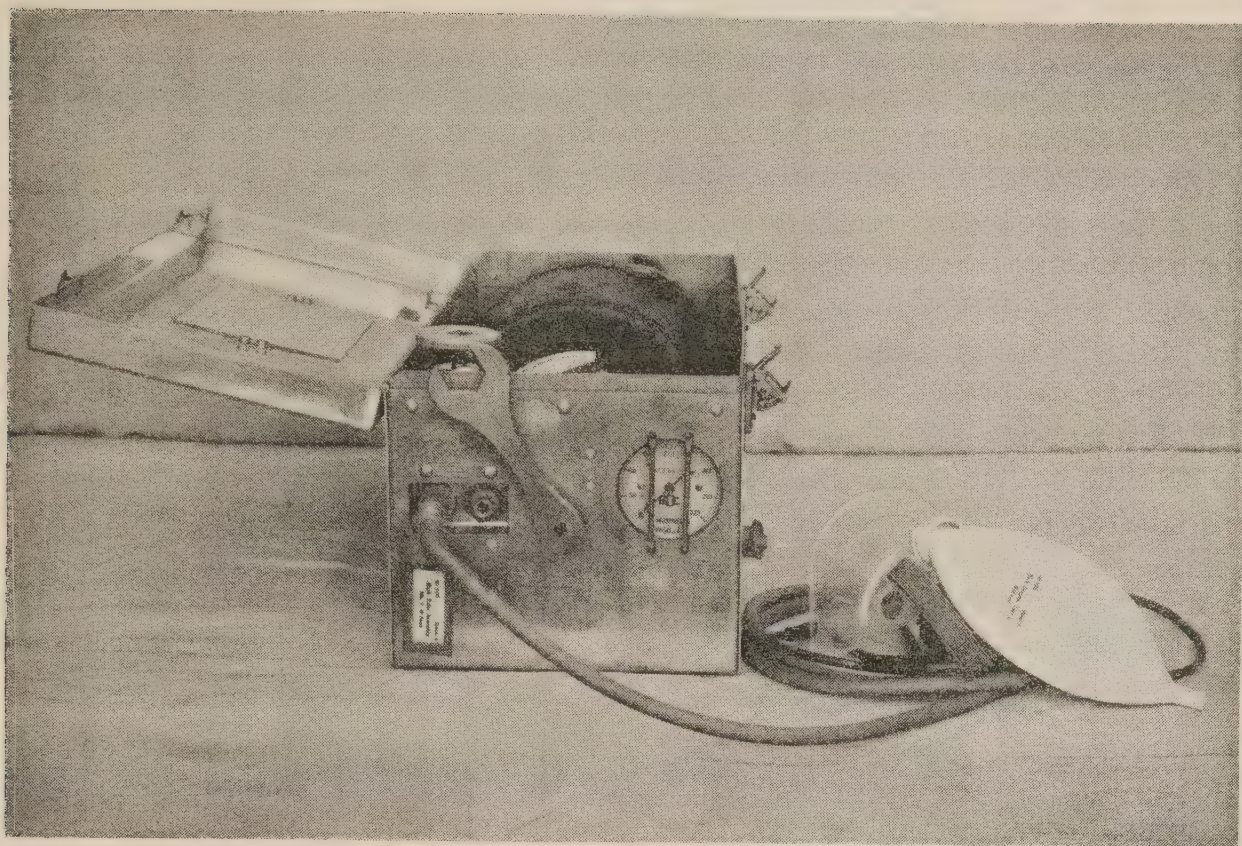
Fig. 4.
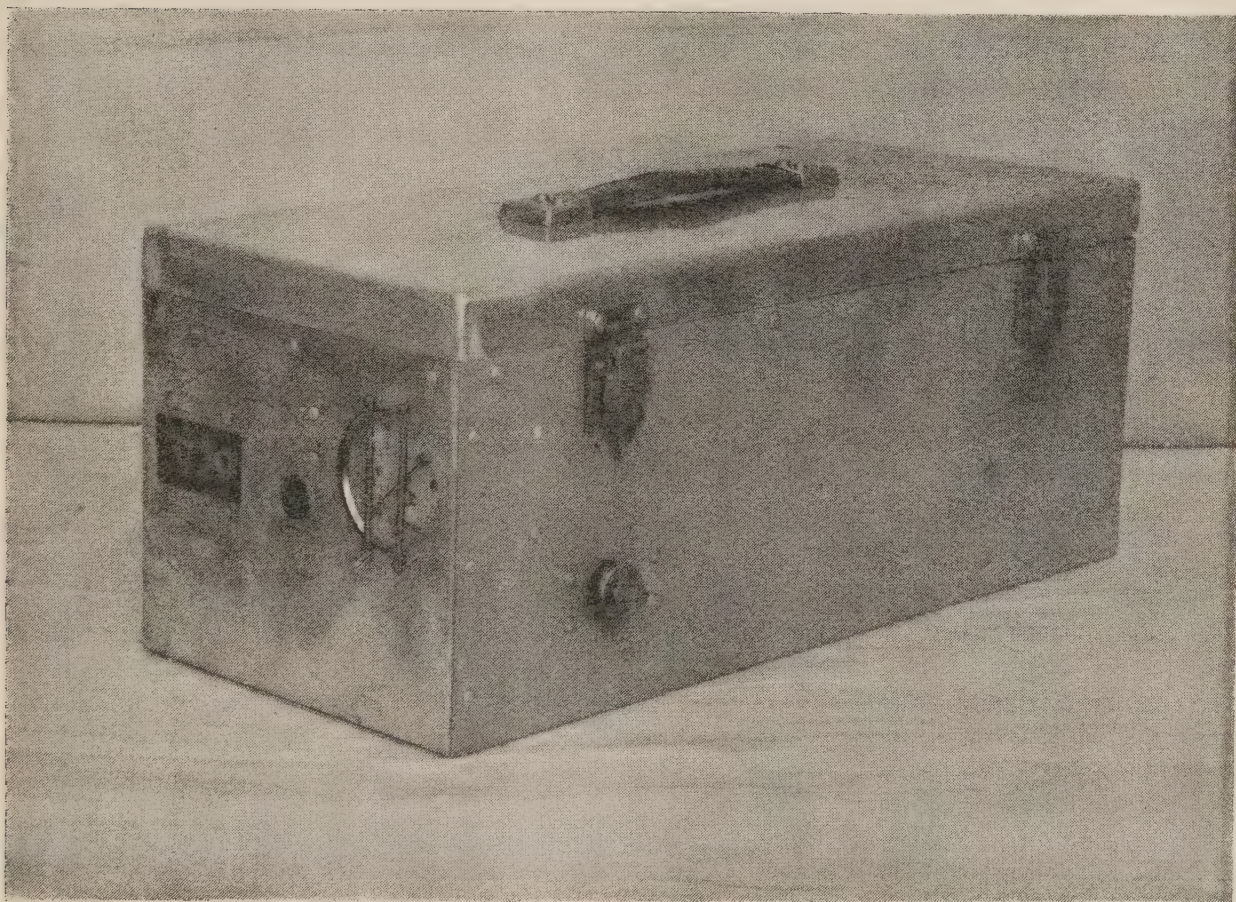
Fig. 5.

Other Methods of Administering Oxygen

266. *Nasal catheters and forked nasal tube.* Oxygen can be administered by means of a catheter passed up the nose, a Jacques rubber No. 9 being suitable. To be effective it is necessary for the tip to be passed up the nostril for a distance of approximately one and a half inches.

A more satisfactory method whereby the oxygen is delivered through two catheters attached to a forked tube is also employed.

In this method the oxygen is conveyed in a rubber tube from the cylinder, fitted with the usual gauge, etc., to a metal tube fixed in a vertical position by means of a brow-band on the front of the patient's head.

The lower end of this tube, which is bifurcated, turns up opposite the nares. Two rubber catheters are attached to these ends, and pass into the nostrils. To get the best results a clear airway is essential.

An alternative form of catheter carrier has been described by Tudor Edwards. In this type the carrier is incorporated in a spectacle frame, and in a modified form the device can be clipped on to the normal spectacle frames if these are already worn.

267. *Oxygen tent.* The use of a tent permits the patient to remain for any length of time in an atmosphere enriched to any desired extent with oxygen with much greater comfort and less restriction than the B.L.B. or other apparatus.

The tent requires skilled attention and is only suitable for use in hospitals. It is made from impervious material which is tucked in all round the bed under the mattress, access to the patient being obtained through wide sleeves in the upper part of the tent. Oxygen is supplied by means of an injector on the cylinder which simultaneously draws air from the tent and passes it through soda lime to remove the carbon dioxide and returns the purified air to the tent. A metal ice-box fitted to the side of the tent cools the interior and moisture is removed by condensation on the box. In an efficiently operated tent the consumption of oxygen is about 3 litres per minute.

268. *Service respirator.* Should cases have to be treated *during* a gas attack, oxygen can be administered by passing it through a wide-bore serum needle thrust either into the facepiece, or into the lower end of the corrugated tube in respirators with this type of construction.

Artificial Respiration.

269. There are two methods of artificial respiration, *i.e.*, manual and mechanical.

(a) Manual. Until recently the most popular method of artificial respiration was the Schafer method. Recently however, this method has been replaced by the *Back Pressure/Arm Lift Method of Holger Nielsen* which is described below.* This was first described in 1932, and recent investigations have shown that this method gives a much greater aeration of the lungs than the Schafer method and would therefore usually be preferred.

In nerve gas poisoning, however, owing to the spasm of the musculature of the shoulder girdle etc., this method may be impracticable and the Schafer method may have to be used.

*Several modifications of this method have been described. The account given here follows that given in the B.R.C.S. First Aid Manual and the R.A.M.C. Training Pamphlet No. 3.

If the shoulder girdle has been injured it would obviously be impracticable to carry out this method. As an alternative for use in cases where the arms or the shoulder girdle have been injured the *Hip Lift/Back Pressure Method*, also described below, has been devised.

(i) ARTIFICIAL RESPIRATION—HOLGER NIELSEN BACK PRESSURE ARM LIFT METHOD

In the Holger Nielsen method of artificial respiration, the chest is compressed (expiration) against the ground by pressure applied to the back, and expanded (inspiration) by raising the arms and taking the weight off the chest. This method ensures good expansion of the lungs and this helps to oxygenate the blood reaching the heart. Quick oxygenation of the heart muscle is essential to recovery of cases needing artificial respiration.

(1) **Lay the casualty face downwards with head turned to one side, arms bent and forehead resting on his hands,** so as to keep mouth and nose free from obstruction.

(2) **Kneel at his head,** placing one knee near the head and the other foot alongside the elbow. From time to time, this position can be altered by changing the kneeling knee, that is, black to white in Fig. 6, (Figs. 6 and 7).

(3) **Place your hands over his shoulder blades,** with thumbs touching on the mid-line and fingers spread out, the arms being kept straight (Figs. 6 and 7).
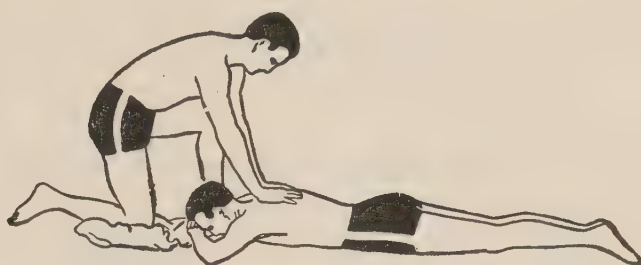
FIG. 6.   FIG. 7.

The Correct Starting Position.
Note the positions of the Rescuer's Knee, Foot, and Hands.

(4) **Bend forward with arms straight** and apply light pressure by the weight of the upper part of your body while steadily counting ' one, two, and three ', in $2\frac{1}{2}$ seconds, to force air out of the lungs (Figs. 8 and 9).
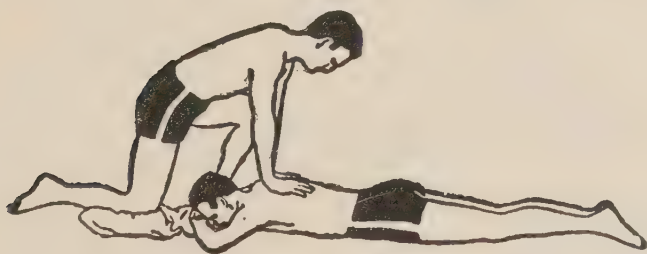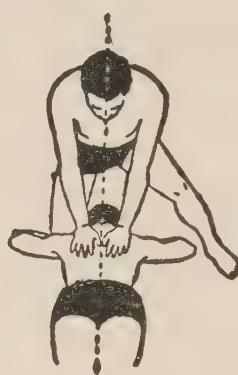
FIG. 8.   FIG. 9.

' One, Two, and Three '. Expiration.

(5) **Release the pressure gradually and slide your hands to just above the elbows of the casualty,** while counting ' four '.

(6) **Raise his arms and shoulders by bending backwards** with your arms straight till you feel resistance and tension, without lifting the chest off the ground, while counting ' five, six, and seven ', in $2\frac{1}{2}$ seconds, to draw air into the lungs (Figs. 10 and 11).

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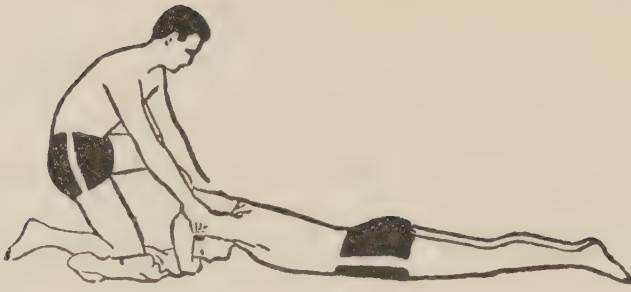
FIG. 10.   FIG. 11.

'Five, Six, and Seven'.   Inspiration.

(7) **Lay his arms down,** then replace your hands on his back as in Figs. 6 and 7, while counting 'eight'.

(8) **Repeat movements** 3 to 7 with rhythmic rocking at the rate of 9 times to the minute, counting as follows:

'One, two and three': with hands on shoulder blades, bend forwards and apply pressure ($2\frac{1}{2}$ seconds);
'Four': slide hands to elbows (1 second);
'Five, six, and seven': bend backwards raising arms and shoulders ($2\frac{1}{2}$ seconds);
'Eight': lay arms down and place your hands on shoulder blades (1 second).

(9) **When breathing is re-established,** carry out arm raising and lowering (6 and 7 above) alone, 12 times to the minute, counting as follows:
'One, two, and three': arm-raising (inspiration, $2\frac{1}{2}$ seconds);
'Four, five, and six': arm-lowering (expiration, $2\frac{1}{2}$ seconds) (Figs. 12 and 13).

FIG. 12.

'One, Two, and Three'.   Inspiration
When Breathing is Re-established.

FIG. 13.

'Four, Five, and Six'.   Expiration.
When Breathing is Re-established.

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(10) **If there are chest injuries,** do the arm raising and lowering procedure only at the rate of 12 times a minute.

(11) **If the arms are injured,** place them by the sides of the body; then do the complete procedure but insert your hands under the casualty's shoulders and raise them for inspiration.

(12) **If arms and chest are both injured,** do arm raising and lowering by inserting your hands under the casualty's shoulders only.

(13) **Apply different degrees of pressure depending on sex and age;** the amount should be just sufficient lightly to compress the chest; the smaller the individual, the less the pressure required.

24-30 lb. for an adult:

12-14 lb. for half-grown children and slender women:

2-4 lb. for infants:

It is advisable to practise these pressures on a spring weighing machine placed about 12″ from the floor.

(ii) HIP LIFT/BACK PRESSURE METHOD

(1) Place the patient face downwards, with face turned to one side and resting on the back of one hand. The other arm is extended forwards.

(2) Kneel with one knee at the level of the hip, straddling the patient and placing the other foot near the opposite hip. (Fig. 14).

FIG. 14.

(3) Place the hands under the patient's hips and raise the pelvis upwards four to six inches. Replace the hips on the ground. (Fig. 15).

FIG. 15.

(4) Transfer your hands to the patient's back just below the shoulder blades, with the fingers spread out, and the thumbs about an inch from the spine. (Fig. 16).

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FIG. 16.

(5) Rock forward, keeping arms straight, and apply steady even pressure down-
wards. Do not use force. (Fig. 17).

FIG. 17.

(6) Rock backwards, release the pressure, then grasp the pelvis and repeat the
cycle about 12 times per minute.

(7) The hip lift causes the patient to breathe in; the back pressure causes the
patient to breathe out.

(b) Mechanical. Artificial respiration by mechanical means may be carried out
by two types of apparatus:—the " Iron Lung " and the Positive Pressure
Resuscitator.

The Iron Lung type of apparatus is obviously quite unsuitable for use in the
field or for provision on a large scale, and is therefore not described here.

The *Positive Pressure Resuscitator* is small and efficient. It consists of a
small hand bellows fitted with a face-piece. The main application of this type
of resuscitator is in nerve gas poisoning where the lungs must be ventilated in
spite of the bronchial constriction.

Artificial respiration. When using the hand bellows the patient should be
supine and the attendant should kneel to the right of the head. His left hand
should encircle the face and hold the face-piece firmly in position (Fig. 18);
the face-piece must fit under the chin. The attendant's other hand should
work the bellows so that the main force is exerted at an angle of 30° to the
horizontal (Fig. 19). This is to ensure that the lower jaw and tongue do not fall
back and obstruct the air-passages. The pressure used should not exceed 20 cm.
of water, since with higher pressures the lungs and stomach may be over-inflated.
The resuscitator illustrated has a safety valve on the face-piece which blows
off at this pressure. Ventilation should be carried out at a rate of 20 to 30 times
a minute and should be continued until the subject is capable of breathing
adequately himself. This will probably occur within 30 minutes, but it must be
remembered that respiration may fail again due to excessive elimination of
carbon dioxide during the artificial ventilatory period; hence the subject should

be kept under observation. During artificial ventilation the stomach will be inflated with air by the resuscitator. This will cause some respiratory embarrassment when normal respiration is re-established. It can be minimized, however, by firm pressure over the stomach, applied intermittently to force the air up the œsophagus. During this manœuvre the patient's head and shoulders should be raised, so as to ensure that it is air and not the liquid contents of the stomach which is forced into the œsophagus. If salivation is excessive while artificial respiration is being carried out, the subject's head should be tilted down periodically to allow the secretions to drain away.

Fig. 18.

Fig. 19.

TABLE I

Summary of Gases, Properties, Methods of Recognition and First Aid

Gas	Classification	How recognized	Effects on the Body	Action to be taken in Gas Attack	First Aid	Remarks
Convulsant Gases 1. Nerve Gas	Semi-persistent. Lethal.	A colourless gas or colourless or dark brown liquid. Quickly recognized by effects.	Tightness of the chest; headache; running nose; contraction of pupils; dimness of vision; nausea and vomiting; sweating; excessive salivation; convulsions; dyspnoea; respiratory failure.	Adjust respirator. Swab liquid from exposed skin.	Give atropine 2 mgm. intramuscularly as soon as possible. Repeat at 15 min. intervals up to total of 10 mgm. or until patient's condition improves. Apply artificial respiration if necessary. Swab off any liquid on the skin.	Speed is vital in treating casualties and atropine must be given as soon as possible.
Vesicant or Blister Gases 2. Mustard Gas	Very persistent. Lethal. Persists from one day to several months depending on temperature, wind etc.	Pale straw to dark brown oily liquid or colourless gas. Smell of garlic or onions. Detector paint, paper and powder change colour.	(a) **Liquid** **Eyes.** Delayed redness followed by closing of the eye and temporary or permanent blindness. **Skin.** No immediate effect. Blisters in 2 hours. Erythema in 2 hours. Blisters 12–24 hours. (b) **Vapour** **Eyes.** Pain and redness in 2–8 hours followed by temporary blindness. **Skin.** Redness, irritation and blisters. Moist parts particularly affected. **Respiratory Passages.** No immediate effects: hoarseness and cough 6–12 hours after exposure. May be followed by bronchopneumonia.	Adjust respirator and anti-gas clothing. Watch detectors for evidence of splash, etc.	Remove outer clothing and any other contaminated clothing. **Liquid** Mop up any visible drops. **Eyes.** Flush out with water immediately for 5 mins. **Skin.** Apply anti-gas ointment No. 6; bleach paste, etc. **Clothes.** After removal, apply ointment or bleach paste to underlying skin, wash skin with soap and water or use solvents. **Vapour** Remove clothing. Wash patient with soap and water. Eye and lung cases should be evacuated as soon as possible. **Blisters.** Treat as burns. Do not apply ointment.	Impervious clothing protects against Mustard Gas, e.g., oilskin, rubber, etc. The respirator protects the respiratory passages and face completely. Do not apply anti-gas ointment to the skin if it is already red or blistered or if it has only been exposed to vapour.

TABLE I—continued

Gas	Classification	How Recognized	Effects on the Body	Action to be taken In Gas Attack	First Aid	Remarks
3. Lewisite	Persistent. Lethal.	Colourless gas or colourless (pure) or brown (crude) liquid. When crude, smell of geraniums. Liquid: stinging sensation on the skin. Vapour: stinging and irritation of the nose. Detector paint, paper and powder change colour.	(a) Vapour. Causes irritation of the nose. Hence respirator may be put on immediately. Skin. Redness in 1 hour may be followed by blistering, less effective on skin than Mustard Gas vapour. (b) Liquid: Eyes. Immediate and grave effect. Severe pain and blepharospasm. Skin. Immediate stinging pain. Blisters occur earlier than with mustard gas. If swallowed in food or water may cause death.	As for Mustard Gas	Apply BAL or anti-gas ointment to skin if unblistered. Give BAL intramuscularly. BAL ointment may be applied to the eyes after they have been irrigated with water (if available). Water is more effective with Lewisite than with Mustard Gas. Clothes should be removed and the underlying skin treated with BAL ointment.	Similar to Mustard Gas but absorbed more quickly and acts more quickly. Anti-gas treatment must therefore be quicker.
Choking Gases						
4. Phosgene	Non-persistent. Lethal.	Colourless gas which may form a white cloud. Smell of musty hay.	Highly lethal. Coughing; lacrimation; choking sensation; dyspnoea; skin leaden-grey in colour. Blue tips of ears and fingers. Cough may cease temporarily to recommence later. Effects develop in 0–24 hours.	Adjust respirator	Evacuate. Complete rest. Warmth and treatment for shock.	
Lacrimatory Gases						
5. C.A.P.	Non-persistent as vapour. Persistent in solid form. Non-lethal.	Greyish crystalline solid. Immediately recognized by its effects. Smell of cheap scented soap.	Stinging pain in eyes. Immediate profuse lacrimation. Blepharospasm. Irritation of exposed skin.	Adjust respirator	Recovery usually rapid without treatment.	
6. B.B.C.	Persistent. May persist for many days.	Pure yellowish brown crystals. As used—brown liquid. Onset of effects. Pungent penetrating smell	Irritates nasal mucosa but not skin, otherwise similar to C.A.P.	Adjust respirator	Recovery usually rapid without treatment.	

TABLE I—continued

Gas	Classification	How Recognized	Effects on the Body	Action to be taken In Gas Attack	First Aid	Remarks
Paralysant Gases						
7. Hydrocyanic Acid	Non-persistent. Lethal.	Colourless gas or volatile liquid. Smell of bitter almonds.	Small amounts of vapour cause giddiness and headache. Larger doses cause unconsciousness and death.	Adjust respirator	Treatment very urgent. Remove from dangerous area. Give cobalt salts to severe cases (1·5 gm of cobalt acetate in 20 ml of water intravenously).	The liquid can be absorbed through the skin. SPEED in treatment is most urgent.
8. Cyanogen Chloride	Non-persistent. Lethal.	Colourless gas or volatile liquid.	Small amounts of vapour cause stinging in the nose, smarting of the eyes, lacrimation and blepharospasm. Larger doses cause symptoms similar to those caused by hydrocyanic acid.	Adjust respirator	Apply artificial respiration. Oxygen inhalations. Warmth and inhalations of amyl nitrite	
9. Carbon Monoxide	Non-persistent. Lethal.	Colourless, odourless gas. Onset of symptoms.	No warning before onset of symptoms. Giddiness, headache and muscular weakness followed by unconsciousness and death.		Remove from dangerous atmosphere. Keep warm: apply artificial respiration with oxygen if available.	First Aid is URGENT. The respirator does not afford any protection.
10. Arsine	Non-persistent. Lethal.	Colourless odourless gas. In high concentrations smell of acetylene or garlic. No warning before onset of symptoms. Special detector papers turn yellow-brown.	Headache and vomiting; loss of colour; muscular weakness; haemoglobinuria; jaundice and progressive anaemia.	Adjust respirator	Urgent artificial respiration. Oxygen inhalations. Blood transfusions. Complete rest.	
Screening Smokes						
11. Hexachlorethane Mixture	Non-persistent. Non-lethal.	White to grey smoke. Sharp stinging nasal sensation.	Heavy concentration irritates eyes, nose and throat.	Adjust respirator	Wash out eyes if affected. Wash skin burns with water.	Concentrations causing symptoms are only likely to occur near the source or after accidental ignition of stocks.
12. Titanium Tetrachloride		White smoke. Sharp stinging nasal sensation.	Liquid burns skin.			
13. Zinc Chloride		White smoke.	May cause nausea and vomiting.			
14. Phosphorus		Burns to white smoke. Nasal irritation.	Burns skin.	Avoid burning particles.	Keep wet with water or cover with copper sulphate Remove particles.	

TABLE II

Effects of War Gases on Food and Water

	Food in Air Tight Containers (including eggs in shell)	Fatty Foods (butter, lard, suet, milk (in open containers) cheese, bacon, fat meat)	Non Fatty Food with high moisture content and crystalline structure (fresh fruit, vegetables, sugar, salt, crushed oats)	Non Fatty Food with low moisture content and amorphous structure (cereals, rice, sago, dried fruits, tea, coffee, flour, bread)
Nerve Gases *Vapour*	No poisonous action	*Condemn*	Condemn unless food can be aired for 48 hours and *then cooked by boiling*	Condemn unless food can be aired for 48 hours and *then*
Liquid	Wash down outer surface—Destroy eggs	Condemn all food contaminated by liquid Nerve Gas		
Mustard Gas *Vapour*	No poisonous action	These absorb and retain mustard gas vapour in surface layers: *Fats:* Remove outer layers to a depth of 1 in. *Milk*—*Condemn*	Unaffected by exposure to mustard gas vapour. Wash down where possible, and air for 48 hours	Absorb mustard gas vapour but release it rapidly on airing. Wash down where possible and air for 48 hours
Liquid	Apply bleach treatment to outer surface, and wash off thoroughly with water. Destroy shell eggs	*Condemn all food contaminated by liquid mustard gas unless it can be processed as advised by higher authority.*		
Nitrogen Blister Gases *Vapour*	No poisonous action	Penetrate more deeply than mustard gas. *Fats:* Remove outer layers to a depth of 1½ inches *Potatoes:* Wash and air 24 hours. *Meat:* Boil for 3 hours in more than one lot of water discarding the water each time.	As for Mustard Gas	
Liquid	As for mustard gas	*Condemn all food contaminated by liquid nitrogen blister gases unless it can be processed as advised by higher authority.*		

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TABLE II—continued

	Food in Air Tight Containers (including eggs in shell)	Fatty Foods (butter, lard, suet, milk (in open containers) cheese, bacon, fat meat)	Non Fatty Food with high moisture content and crystalline structure (fresh fruit, vegetables, sugar, salt, crushed oats)	Non Fatty Food with low moisture content and amorphous structure (cereals, rice, sago, dried fruits, tea, coffee, flour, bread)
Arsenical Blister Gases (*e.g.*, Lewisite) *Vapour*	No poisonous action			
Liquid	Wash down outer surface. Destroy shell eggs.	Foodstuffs contaminated by liquid lewisite may contain dangerous concentrations of arsenic. As a general rule such foodstuffs should be *condemned*		
Phosgene. Diphosgene. Chloropicrin.	No poisonous action	Very little effect. Expose to air for 24 hours. Food is then ready for human consumption.	As for Fatty Foods	As for Fatty Foods. Infusions of tea and coffee may have a slightly bitter taste. Flour does not bake well after exposure but if mixed with uncontaminated flour gives satisfactory results.
Nose Gases	No poisonous action	These gases are arsenical compounds. Food is liable to be contaminated to a dangerous level and as a general rule should be condemned.		
Tear Gases	No poisonous action	Render food unpalatable but not toxic. Contamination by Tear Gas only does not make food unfit for human consumption. Contamination by liquid B.B.C. may render the food so unpalatable that it will have to be destroyed. *WARNING:* Remember that Tear Gas may be mixed with other more poisonous gases.		
Others, *e.g.*, Arsine or HCN		Unlikely to affect foods in the concentrations that would be achieved in the field.		

*The technical expert may be the Officer-in-Charge Mobile Pathology Laboratory, Officer-in-Charge Mobile Hygiene Laboratory, a R.A.S.C. Chemist, a Technical Officer Special Weapons or any other analytical chemist.

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COLOURED PLATES

The following plates illustrate the effects of poisoning by mustard gas. Plate II is taken from a paper by Dr. Ida Mann, F.R.C.S. and Dr. B. D. Pullinger in the *Proceedings of the Royal Society of Medicine*, for January 1942 (Vol. 35, No. 3), and is reproduced by courtesy of the Honorary Editors of that journal. The other plates were supplied from the records of the Chemical Defence Experimental Establishment by arrangement with the Medical Superintendent. The copyright of all the plates is reserved.

Plate I. Effect of Mustard Gas Vapour on the Face.

Typical appearance of the face 24 hours after exposure to mustard gas vapour (*see* para. 96).

(*Certified correct R. M. Barnes, Capt., for D. Path.*)

Plate II. Effects of Mustard Gas on the Eye (*see* para. 98).

FIG. 1.   Severe lesion three weeks after exposure to mustard gas vapour, showing a small patch of hyperæmia in the palpebral conjunctiva (A), a triangular white necrotic patch at the inner side of the limbus (B), and a slight corneal haze in the palpebral aperture (C).

FIG. 2.   Severe lesion after exposure to a spray of liquid mustard gas, showing subconjunctival hæmorrhages, dilated thrombosed vessels and partial destruction of limbal capillaries in exposed areas which were formerly necrotic and opaque ; and in the cornea, epithelial bedewing with œdema of the substantia propria below.

FIG. 3.   Severe lesion five and a half weeks after exposure to a spray of mustard gas (the same eye as Fig. 2), showing an area of necrosis on the palpebral conjunctiva, hæmorrhages in the ocular conjunctiva with an area of necrosis at the limbus below, some bedewing of the corneal epithelium and clear substantia propria. (Stage of subsidence of primary œdema).

FIG. 4.   Severe lesion eight weeks after exposure to a spray of mustard gas (the same eye as Figs. 2 and 3) showing partial vascularization of the necrotic patch in the palpebral conjunctiva, hyperæmia of the ocular conjunctiva around and in the former necrotic white area, reappearance of corneal œdema below, and vascular invasion of the cornea with thrombosis and hæmorrhage in this region.

FIGS. 5 AND 6.   Very severe lesions (so-called delayed keratitis) in both eyes, 23 years after exposure to mustard gas and 13 years after the first attack of delayed ulceration.

R.E. Fibrous plaque of ocular conjunctiva at outer side ; abnormal varicose conjunctival vessels in outer and lower part ; gross scarring of lower part of cornea and limbus.   Posterior synechiæ.

L.E. Triangular fibrous plaques at inner and outer side of limbus, tortuous and varicose conjunctival vessels, dense corneal scarring with vascularization and posterior synechiæ.

PLATE II

From A Study of Mustard Gas Lesions in the Eyes of Rabbits and Men.
By Ida Mann and B. D. Pullinger

(*Proc. R. Soc. Med.*, 1942, **35**, 229).

Plate III. Effect of Liquid Mustard Gas on the Skin (24 hours).

Blister appearing on the skin 24 hours after exposure to liquid mustard gas (*see* para. 99).

(This could also happen from want of care in decontamination).

Plate IV.   Effect of Liquid Mustard Gas on the Skin (5 days).

Destruction of outer layers of the skin following blister formation five days after exposure to liquid mustard gas (*see* para. 99).

Plate V. Effect of Liquid Mustard Gas on the Skin (7 days).

Scarring and coppery pigmentation of the skin seven days after exposure to liquid mustard gas (*see* para. 99).

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